THE APPROACH TO CARDIOLOGY

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THE APPROACH TO CARDIOLOGY

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EXAMINER IN MEDICINE TO THE UNIVERSITIES OF CAMBRIDGE, EDINBURGH DURHAM ABERDEEN AND SHEFFIELD

WITH A FOREWORD BY

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PREFACE

THE transition from the pre clinical to the clinical period of training is difficult for the student, he is at a loss to know how to apply knowledge gained in the laboratory to work in the wards He arrives full of enthusiasm, but is apt to become discouraged To bridge this gap we instituted, in Manchester in 1943, an introductory course of lectures which the student attended during his first six months in hospital This course, the main purpose of which was to illustrate the application of the pre-clinical sciences to clinical work, consisted of seventy lectures in the following subjects Pathology (11) Medicine (23), Surgery (24), Obstetrics and Gynaecology (6), and Child Health (6) This book is based on lectures forming part of that course

Concurrently with the first three months' lectures, the *students received elementary practical instruction in Patho*logy and Bacteriology, and in the methods of physical examination of patients, for the latter the class was divided into groups of twelve During the second three months they held their first appointment as medical clerk or surgical dresser

A close liaison was established between the clinical and pre clinical departments, by attaching junior members of the clinical staff to the departments of anatomy and physiology, an arrangement greatly appreciated by students, both because it introduced them to clinical problems at an earlier stage of their course and because there was a sense of continuity when they met their pre clinical teachers in the wards

My experience as an examiner has led me to believe that there is a danger of students being so overburdened with detailed factual knowledge that they can no longer see the wood for the trees In this monograph I have made no

attempt to cover any part of the field, but have merely tried to illustrate some of the fundamental general principles of cardiology in relation to the pre-clinical sciences

I am indebted to many of my colleagues for their helpful criticism, especially to Dr E Duff Gray, who has also kindly provided the radiograms, to Professor G A G Mitchell, to Professor R V Christie, and to my brother, Professor Edwin Bramwell I should also like to express my keen appreciation of the never-failing help and consideration I have received from Mr G T Hollis of the Oxford University Press

I am grateful to the publishers of *Heart*, the *Quarterly Journal of Medicine*, the *British Heart Journal*, *the Lancet*, and the *British Medical Journal* for permission to reproduce illustrations from my original papers which they published, to Messrs Arnold & Co for illustrations from my book *Heart Disease*, to Sir John Parkinson for Figure 10, to the Department of Roentgenology of the Johns Hopkins Hospital for Figure 15, to the medical artist and to the Photographic Department of the Manchester Royal Infirmary for some of the diagrams

C B

MANCHESTER
January 1951

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FOREWORD

THE Journal of Physiology in 1911 contained a paper by J Crighton Bramwell and Keith Lucas on the refractory period of nerve. Their experiments were made in a cellar at Cambridge where Adrian also worked on the other side of a partition my instruments consorted with Gowland Hopkins's rats The drains did not operate very well and the flaring gas burners were bewildering, but I have never ceased to be grateful for the intellectual companionship of that cellar From 1920 to 1923 another cellar, this time in Manchester, brought Bramwell and me together again in experiments on the pulse wave and the elasticity of arteries There, and in his company occasionally at the Royal Infirmary, I learnt a little of the principles on which the methods and ideas of physiology can be employed in clinical medicine The memory of our collaboration makes it a very special pleasure to give an affectionate send-off to a book in which those principles are so evident, and so fruitfully applied

A V HILL

UNIVERSITY COLLEGE, LONDON
29 January 1951

CHAPTER I

THE CLINICAL APPROACH

THE transition from pre-clinical to clinical work is almost as sudden and as startling as that from chrysalis to butterfly When the student leaves the laboratory and enters the ward, he finds himself in a completely strange environment, the problems with which he is confronted are so different from those which he had met hitherto that it is difficult to see how to apply to them the knowledge he has previously acquired

THE ART OF MEDICINE

Between the pre-clinical and the clinical studies a great gulf is fixed, and to help to bridge that gulf is the purpose of this monograph The pre-clinical subjects, especially physiology, constitute one of the essential foundations on which to build knowledge of clinical medicine I say *one* of the foundations because clinical medicine is far more than mere applied physiology Body and the mind are inseparable, they constantly react upon one another and, to become a competent clinician, it is essential to appreciate this fact Men with brilliant academic records who, as students, carried all before them, sometimes make very poor doctors, because they think only of the disease and ignore the patient Our job is to treat John Brown and not merely 'a case' of heart disease Bill Smith may be suffering from exactly the same disease as John Brown, but the treatment he requires may be entirely different, because the clinical picture in every case is modified by the psychological background of the patient Those who have had the task of coaching a team realize that each man has to be treated differently, some lack self-confidence and require

encouragement, while others are over confident and have continually to be reminded of their limitations The same is true of patients, some belittle their suffering, others exaggerate it The neurotic, introspective individual arrives at the consulting-room armed with a list of his symptoms, he has written them down for fear his memory should play him false and fail to do justice to the seriousness of his complaint The very fact that he has done so gives the physician a clue to his temperament In striking contrast to the neurotic is the man who would appear to be almost ashamed of troubling the doctor and apologizes for wasting his time He sits with sealed lips and one has to drag the story out of him

Most patients consult a doctor either on account of some symptom which is producing an actual physical disability, or on account of symptoms which, though in themselves trivial, alarm him, because he believes that they are the first signs of a serious or perhaps fatal malady He broods over his troubles by day, they rob him of his sleep by night, and he develops what we call an 'anxiety state' If we take the bull by the horns and ask him for his own explanation of his symptoms, he may even confess, 'I thought, doctor, it might be cancer' Patients do not realize what an important part psychological factors play in the production of ill health, they naturally attribute all their symptoms to physical causes

Apart from these anxiety states, a patient may be unhappy in his home life or in his work, he may be unable to adapt himself to his environment Such maladjustment often gives rise to physical manifestations of ill health mimicking those of organic disease, especially in war-time, when so many square pegs are fitted into round holes

Another factor that plays a large part in the production of symptoms is physical unfitness The athlete, training for a contest, requires plenty of exercise and plenty of sleep, he has to forgo tobacco and alcohol in order to get fit, for, when

he is out of condition owing to overwork, oversmoking, or any other cause, he becomes breathless on exertion and manifests other symptoms not unlike those of heart disease The same is true of patients convalescent from an acute illness, such as influenza Hence, in assessing the factors responsible for a patient's disability, it is important to remember that every case of organic disease is likely to be complicated to a greater or lesser extent by both physical unfitness and psychoneurosis and that both these complications call for treatment

Consider the case of a patient suffering from angina pec toris Disease of the coronary arteries is a common basis for this syndrome, but the fact that the patient is overweight, that he smokes too much, or that his muscles are flabby because he avoids exercise for fear of an attack, all increase his disability, while anxiety, lest sudden death may prevent his making adequate provision for his dependants, adds to his burden

To take another example, a supposedly healthy young man presents himself for life insurance, and is turned down on account of a heart murmur He consults his family doctor who confirms the presence of the murmur and tells him, quite rightly, that it is due to a slight abnormality which has been present from birth, but he fails to impress on him the fact that it is of no importance and is never likely to cause trouble Never having been ill in his life, the young man is naturally worried He decides to give up sport and to conserve his energy, with the result that he soon begins to put on weight and to get out of training He notices that he is becoming breathless going uphill and is sometimes conscious of his heart beating, a thing he had never noticed before These symptoms he naturally attributes to heart disease He accordingly restricts his activities still further He puts on more weight and becomes more breathless, and eventually decides

to consult a specialist who assures him that his heart lesion may be completely disregarded, but has great difficulty in eradicating the neurosis which by now is deep-rooted The cause of the disability in such a case is not the structural lesion, but the neurosis, and it is vital to appreciate this fact, for, whereas structural damage is generally beyond our control, physical unfitness and psychoneurosis are amenable to treatment

When the disability is entirely attributable to physical unfitness and psychological factors, without any organic basis, we say that the patient is suffering from 'Effort Syndrome', or as it is called in America 'Neuro-circulatory Asthenia' In this condition the response of the circulatory and respiratory mechanisms to exertion is excessive, the patient's ability to perform muscular work is decreased and mild exertion is sufficient to cause breathlessness, palpitation, and other symptoms, the increase in heart-rate after exercise is greater than in health and it takes longer for the pulse to return to its resting rate

One may compare the various factors which give rise to symptoms of circulatory insufficiency to a spectrum (Table I)

TABLE I

Infra red	*Visible spectrum*		*Ultra violet*
Organic	Neuro circulatory asthenia		Neurosis
	Physical	Mental	

In the centre is the visible spectrum representing neuro-circulatory asthenia, at the red end are the aetiological factors to which I have referred as responsible for producing physical unfitness, at the violet end, the psychogenic factors In the infra-red region lie the organic diseases, while, at the ultra-violet, are the pure anxiety neuroses

DIAGNOSIS

The practice of medicine consists of diagnosis, treatment, and prognosis In clinical teaching diagnosis takes prece-

dence, and rightly so, for it is the keystone of the arch, but to the patient it is the least important of the triad The questions uppermost in his mind are 'Is my condition serious?' 'When shall I be fit to return to my work?' 'Will the present illness lead to any permanent disability?' and 'What treatment will help me to get well?' By diagnosis I mean, not merely giving a name to the disease, but rather summarizing the conclusions which one draws from a critical survey of the clinical history and the physical examination

In a case of heart disease the diagnosis may be considered under the following headings

1 What structural changes are present?
2 Is there any disorder of function?
3 What is the underlying cause of the condition?
4 What is the extent of the disability?
5 What is the patient's reaction to his disease?

From the patient's point of view—and a good doctor always looks at things from the patient's point of view—the most important aspect of diagnosis is the extent of his disability and his consequent incapacity, in other words the functional efficiency of his circulatory mechanism

It is convenient to make an arbitary division of disability into different grades, which will necessarily vary in accordance with the nature of the patient's occupation and mode of life The following are the grades suggested by the American Heart Association

Class I No limitation of physical activity
II Slight limitation of physical activity
III Considerable limitation of physical activity
IV Unable to carry on any physical activity without discomfort Symptoms of cardiac insufficiency even at rest

The point which I wish particularly to emphasize is that

the disability is a *relative* and not an absolute quantity You would never dream of comparing a destroyer with a battleship, though both are highly efficient pieces of mechanism Similarly a patient's disability varies in accordance with his mode of life An unskilled labourer may be prevented from earning his living by a cardiac lesion which would cause little inconvenience to a bank clerk Given the same disability, the prognosis for the manual worker and the brain worker is very different, though the brain worker has his own special troubles to contend with, the so-called 'Stress diseases' like coronary thrombosis and duodenal ulcer The extent of a patient's disability varies with the degree of impairment of the efficiency of his circulatory mechanism or, expressed in terms of achievement, inversely with his tolerance for exercise

Each of the other three aspects of diagnosis to which I have referred has at one time dominated the clinical picture the anatomical diagnosis is concerned with structural lesions, such as enlargement of the heart or disease of the heart valves, the physiological diagnosis with disorders of the circulatory mechanism, such as high blood-pressure or irregular heart action and the aetiological diagnosis with the pathology of the condition—is it inflammatory, degenerative, or due to some congenital defect of development?

Diagnosis rests on a twofold foundation—the clinical history and the physical examination, the former is concerned with symptoms, the latter with signs To take an adequate history is far more difficult than to make an adequate physical examination I would expect a good house physician to elicit the physical signs, but it requires long clinical experience to obtain, and to assess at its true value, all the evidence which can be got from interrogating the patient The beginner must learn the art of cross-examination In order to obtain reliable evidence it is important that the physician should avoid suggesting to the patient, however indirectly,

the answer to his questions With this in mind, he should, as far as possible, avoid leading questions (i e questions which can be answered by 'yes' or 'no') Instead of asking a patient whether his pain is situated in a certain region, he should inquire 'Where is your pain?', but with an un-co-operative or stupid patient one may eventually be compelled to resort to leading questions The good clinician, like the good chairman of committee, knows just when to apply the closure he saves time by eliminating irrelevant evidence The novice, on the other hand, is afraid to stop a garrulous patient, for fear of missing some essential point, and lets him ramble on indefinitely

Symptoms result from some disturbance of function They may, or may not, be associated with recognizable structural changes in the body Disorders of function often appear be-before any structural change can be detected and, for that reason, it is important to recognize their significance, since it is in its early stages that disease is most easily arrested

Some symptoms—pain for example—are purely subjective, we have no direct means of estimating their severity Some, such as breathlessness, are not merely distressing to the patient, but apparent to the observer The term 'physical signs' is best reserved for the findings revealed on examination, by inspection, palpation, percussion, auscultation, &c

PHYSICAL METHODS

In the examination of patients there is a danger that the methods we employ may become stereotyped

The orthodox teaching in the past has been to record the findings on physical examination under four headings—inspection, palpation, percussion, and auscultation The beginner is taught to apply them in a routine manner, and is apt to lose sight of the fact that they are merely the means to an end Admittedly it is essential that he should acquire

proficiency in technique, but, once that has been mastered, he should not apply these methods blindly, but should ask himself the question, 'What is my objective in this particular case and how can I best attain it?'

For every investigation which the clinician has to make there is a method of choice, that is to say, a method which yields the most reliable information In the hands of the specialist, this is the method which should, whenever possible, be employed, to the exclusion of all others This method, however, may involve the use of elaborate apparatus which renders it inapplicable in general practice, and it is important that we should consider the problem of diagnosis from the view-point of the general practitioner as well as from that of the cardiologist Let us therefore ask ourselves two questions (1) 'How much information can the experienced general practitioner be expected to elicit by his unaided senses, supplemented only by instruments he habitually uses, such as the stethoscope and the sphygmomanometer?' and (2) 'What further light can the specialist throw on the diagnosis by employing, in addition, the more elaborate methods of investigation such as radiography and electrocardiography?'

In the case of the simpler physical methods, such as palpation and percussion, though both may be capable of yielding information on a particular point, the one may be preferable to the other *For example, if one excludes radiology, the* most accurate guide to the position of the left heart border is the apical cardiac impulse, as localized by palpation When, therefore, the impulse can be accurately localized, it is both unnecessary and irrelevant to say that the dullness on percussion extends to a certain point, since this method is less reliable than palpation, and by palpation we have got all the information we require percussion is redundant Similarly with inspection, the apical impulse may be seen in such and

such a place, but its position is more accurately determined by palpation In this case, inspection is merely a guide to palpation

Finally, we must consider critically whether the various physical methods we use in the diagnosis of disease really do merit the confidence we place in them and are capable of yielding accurately the information we require, above all, we must know their limitations

TREATMENT

One of the things which strikes the student when he leaves the laboratory and starts work in the wards is the difficulty of obtaining adequate controls for clinical experiments At first it may seem that the clinician is a loose thinker who is apt to base conclusions on insufficient premisses The problems of chemistry and physics are so much more clearly defined than those with which the clinical investigator has to deal they are not complicated by the psychological, economic, and other extraneous factors, which add greatly to the difficulty in arriving at conclusions, and limitations unknown to the pure scientist are imposed upon us by the fact that we are dealing with human beings and must never do anything which might prove detrimental to our patients

The aim of research, whether in the laboratory or at the bed-side, is to make a permanent contribution to the advancement of knowledge, the investigator must satisfy himself that the evidence on which his conclusions are based is beyond reproach The practitioner of medicine, however, has a duty to his patients He has to endeavour to relieve suffering and to prolong life by whatever means are at his disposal Such means, although effective, may be purely empirical, but to refuse to employ them because he does not understand how they work would be more than foolish, it would be negligent

It is often extremely difficult, sometimes impossible, to be sure that the improvement in a patient's condition is attributable *to the particular drug or method of treatment employed* Rest and skilled nursing alone work marvels in some cases of heart failure and, in attempting to assess the effect of drugs scientifically, it is desirable to postpone their administration until the full beneficial effect of rest has been observed, for it is only then that we can estimate with any degree of certainty their value When, however, a patient is admitted to hospital critically ill, all therapeutic measures likely to help must be employed at once, delay might endanger life As doctors we are entrusted with the care of human life It is a great responsibility Never, must scientific enthusiasm for the advancement of knowledge tempt us to do anything which might prejudice a patient's chance of recovery

I have already referred to the influence of mind over body This introduces a further complication in assessing the results of treatment Certain drugs are known to have a beneficial effect in certain diseases, they are called specifics, as, for example, salicylates in acute rheumatism, mercury in syphilis, or quinine in malaria Other drugs, such as purgatives and hypnotics, are given to correct a particular disorder of function, but it is important to remember that this is only a subsidiary line of attack, the primary objective being to cure the disease responsible for these symptoms Unfortunately there are many conditions for which we still have no specific remedy, and the benefit for which medicinal treatment may get the credit in such cases is in fact often attributable to psychological rather than to physical action Some 'patent medicines' contain potent drugs and differ only from the equivalent pharmacopoeial preparations in being more expensive, but others are completely inert Nevertheless, skilful advertisement may win for the latter an undeserved reputation owing to the credulity of the public By credulity I mean

willingness to accept as facts statements which are not properly authenticated

A therapeutic difficulty frequently encountered in the practice of our profession is inability of the patient to follow the advice one would like to give him It may be desirable, in the interests of health, for a man to retire, but he may not be able to afford to do so, and then we must be content with a compromise, but, even if his financial circumstances are adequate, he may have no hobbies and, if you take away his work, life will no longer be worth living, we want to enable him to live, not merely to exist

'Social Medicine' is concerned with those economic and other factors which play such an important part in the maintenance of health The Hospital Almoner is able to keep the physician informed of the patient's home conditions, and to help the patient to overcome his difficulties in endeavouring to implement the advice he has been given Thus she provides the lubricant which enables the wheels of therapeutics to revolve, but in private practice every doctor is his own almoner and must make himself familiar with his patient's circumstances, cope with his worries, and be sympathetic even with his idiosyncrasies

CHAPTER II

CARDIAC ENLARGEMENT

CARDIOMETRY

'Is the heart enlarged?' This is the first question we have to answer in making an anatomical diagnosis, for an *enlarged heart is almost always a diseased heart* What we would like to be able to do is to weigh the heart and to express the heart-weight as a percentage of the body-weight, thus obtaining what is known as the *heart ratio*

This ratio is fairly constant in each species of animal, but varies in different species The smallest mammals all have relatively large hearts on account of their high metabolic rate Excluding them, the remainder of the mammalian kingdom may be divided into two classes (Table II)—those which are and those which are not capable of prolonged and severe exertion The athletic animals, such as the deer or the wolf, have a heart ratio of about 1 0 whereas the sedentary animals such as the rabbit or the hedge-hog have a heart ratio of under 0 5

TABLE II

(Modified from Clark, 1927)

Animals with high heart ratios		*Animals with low heart ratios*	
Animal	*Heart ratio*	*Animal*	*Heart ratio*
Deer	1 15	Rabbit (wild)	0 27
Badger	1 08	(tame)	0 3
Wolf	1 02	Field vole	0 37
Weasel	0 94	Hedgehog	0 38
Stoat	0 94	Wild boar	0 39
Fox	*0 92*	*Norwegian rat*	0 4
Seal	0 92	Sheep (domestic)	0 42
Red deer	0 90	Guinea pig	0 42
Hare	0 87	Domestic cat	0 46
Dog	0 85	Domestic ox	0 49

$$\left(\text{Heart ratio} = \frac{\text{Heart wt} \times 100}{\text{Body wt}}\right)$$

Taking 300 gm as the average heart-weight of a man of 70 kilos, the heart ratio in man works out at 0 43 It is rather humiliating to find that, judged by this standard, from the athletic point of view, we are only on a par with the sheep and the guinea-pig! But man, I suppose, must be regarded as a domestic animal, and usually the heart ratio in domestic animals is lower than in wild animals of the same species The heart ratio of the wild duck is 1 06 while that of the tame duck is only 0 63 Similarly the heart ratio of the game cock is 1 03 while that of the rooster is 0 68

TABLE III

(Clark, 1927)

Domestic animals		*Wild animals*	
Animal	*Heart ratio*	*Animal*	*Heart ratio*
Barndoor cock	0 68	Game cock	1 03
Tame duck	0 63	Wild duck	1 06

Unfortunately it is not possible to weigh the heart during life, and we have to be content to measure its size and compare this with the size of the body The simplest comparison to make is between the width of the heart shadow and the width of the chest, this we speak of as the '*Cardio-thoracic ratio*' (Fig 1) Normally the heart width is slightly less than half the chest width, hence a cardio-thoracic ratio of less than 2 0[1] suggests that the heart is enlarged, but since the heart is a three-dimensional organ, the cardio-thoracic ratio being a measurement in one plane only, is not capable of giving us more than a rough idea of its size

An alternative method is to measure the area of the heart shadow and to compare this with the area of the chest This method should give more accurate results since two dimensions are involved, but it is laborious and is subject to two sources of error for it is often difficult to define the point

[1] Some cardiologists express this ratio as heart chest instead of chest heart in which case 0 5 is upper normal limit

at which the shadow of the heart joins that of the great vessels, and it is impossible to obtain an accurate outline of the lower surface of the heart where its shadow merges with that of the liver (Fig 7)

Nylin and his colleagues in Stockholm (1939) have devised a method of obtaining a three-dimensional measurement of

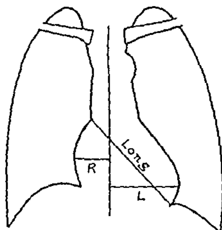


FIG 1 Orthodiagram of typical normal heart

heart volume, by taking simultaneous radiograms at right angles to one another, the one postero anterior and the other lateral A somewhat similar technique has been adopted by Benedetti and Bollini in Bologna (1935), but these methods are adapted for research rather than for routine work

The cardio-thoracic ratio varies with the body-build, in short thick-set people (Fig 2), the diaphragm is high and the chest is shallow, consequently the heart lies transversely, and the cardio-thoracic ratio is high, while in tall, thin people, with a long narrow thorax (Fig 3), the heart lies more vertically and the cardio-thoracic ratio is low Thus in attempting to assess cardiac enlargement from the cardio-thoracic ratio it is essential to take into account the body-build of the patient

Hodges and Eyster (1926) worked out statistically a formula for predicting the transverse diameter of the heart from

weight and height. This predicted value is a more reliable guide to cardiac enlargement than the cardio-thoracic ratio. Bedford and Treadgold (1931) found that in 86 per cent. of

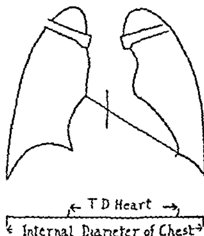


FIG 2 Orthodiagram of transversely disposed heart

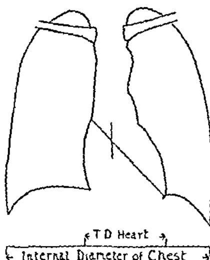


FIG 3 Orthodiagram of vertically disposed heart

a series of 116 healthy adult males the observed transverse cardiac diameter fell within $\pm$ 10 per cent of the predicted value

Two special modifications of X-ray technique known re-

spectively as teleradiography and orthodiagraphy are used in measuring the heart in order to minimize distortion from divergence of the rays (Fig 4)

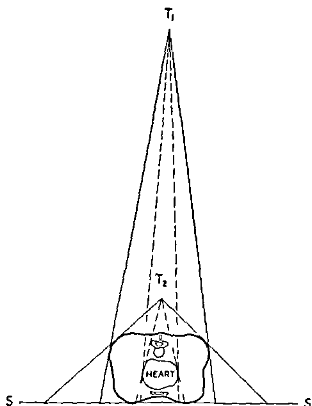


FIG 4 Diagram to illustrate principle of teleradiography When X ray tube (T_1) is 6 feet from screen (*S S*) rays are less divergent than when it is close to body (T_2) and the error of measurement is correspondingly reduced

PALPATION AND PERCUSSION

Before the introduction of X-rays we had to rely on our unaided senses to determine the size of the heart and, in practice, we still often have to do so Percussion is a very unreliable guide to heart size, because the area of cardiac dullness is greatly modified by the structures which intervene between the heart and the surface of the body, the muscular and fatty tissues of the chest wall, and above all by the varying

extent to which the heart is overlapped by resonant aerated lung tissue It is only in children and thin adults that one can rely on percussion Palpation is more helpful In many cases it enables one to locate the position of the apical cardiac impulse, but it is only when this impulse is a well-defined thrust which is strictly localized that it is a reliable guide to the position of the left heart border This is a very important fact which is not sufficiently appreciated If the apical impulse cannot be accurately located, we must generally be prepared to admit that, without the help of X-rays, we cannot say whether the heart is, or is not, enlarged

Cardiac enlargement is assessed by comparing the size of the heart with the size of the chest Radiologically we say that the heart is enlarged if the transverse diameter of the heart is greater than half the transverse diameter of the chest, clinically we say that the heart is enlarged to the left if the apical cardiac impulse is situated beyond the mid-clavicular line, but before arriving at such a conclusion we must be certain that the heart is not merely displaced Gross displacement by lesions such as a right-sided pleural effusion or a pneumothorax is easily recognized, but slight displacement due to slight lateral spinal curvature is very apt to be mistaken for enlargement

Depression of the sternum may cause either displacement of the heart to the left or compression of the heart in the antero-posterior plane with a corresponding increase in its transverse diameter

During the war many recruits were referred to me on account of apparent cardiac enlargement, for which there was no obvious cause In most cases X-ray examination did not confirm the presence of enlargement Though occasionally leftward displacement of the apical impulse was accounted for by spinal curvature or by other causes, I am convinced that, in the majority of cases, the error was due to the

examiner basing his opinion on an impulse which was diffuse and could not be accurately localized

I said that an enlarged heart is almost always a diseased heart There is one important exception The hearts of athletes who indulge in prolonged and severe exertion may

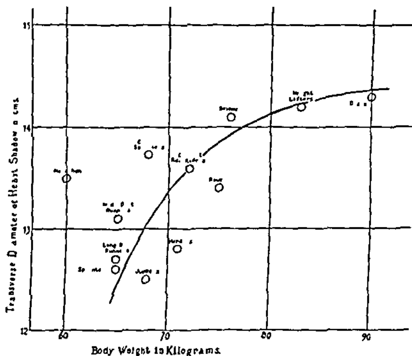


FIG 5 Relation of heart size to body weight in various groups of Olympic athletes

be distinctly bigger than the average, but it would be wrong to consider their hearts diseased, for they are more, not less, efficient than the average

At the Olympic Games in Amsterdam in 1928, Reginald Ellis and I examined over 200 athletes who participated in various types of sport and we compared the average size of the heart with the body-weight in each group When plotted as a graph (Fig 5) these values all fall in the vicinity of a mean curve There is, however, one exception, namely, the group of Marathon runners, whose hearts are unduly large

In fact, comparing Marathon runners with other runners we found that not only were their hearts relatively but absolutely larger, in spite of the fact that they were smaller men, their average body-weight being only 60 kilos

Our finding of cardiac enlargement in Marathon runners agrees with that of most previous workers, but are we justified in regarding this enlargement as entirely attributable to their training? In so doing may we not be putting the cart before the horse? The Marathon runner needs a big heart Doubtless his heart hypertrophies as the result of prolonged training, but it seems possible that there is another factor concerned May it not be that men who happen to have hearts somewhat bigger than the average make the best Marathon runners? They start life with a natural advantage If this be so, it is not the Marathon running that makes the heart big, but the big heart that makes the Marathon runner Another feature peculiar to the Marathon runner is that his resting pulse-rate is generally well below 60 This enables him to increase his cardiac output without an undue increase in heart-rate

In this connexion it is interesting to refer to two recruits with big hearts whom I examined during the war, they were youths of 19 and 20 respectively, both were keen athletes In both these lads the sitting pulse-rate was only 50, a degree of bradycardia very uncommon at that age This association of cardiac enlargement with bradycardia in young athletes is comparable with what we found in Marathon runners who were older men, their average age being 27

Some perfectly healthy people have very small hearts, one of the smallest hearts in our whole series of Olympic athletes was in the winner of the 100 and 200 metres

From what I have said about the cardio-thoracic ratio it will be obvious that it is impossible by this measurement to recognize with certainty the lesser degrees of cardiac

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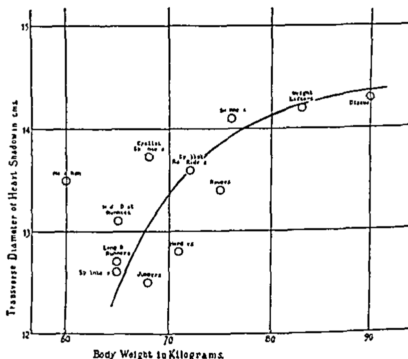


Fig 5 Relation of heart size to body weight in various groups of Olympic athletes

be distinctly bigger than the average, but it would be wrong to consider their hearts diseased, for they are more, not less, efficient than the average

At the Olympic Games in Amsterdam in 1928, Reginald Ellis and I examined over 200 athletes who participated in various types of sport and we compared the average size of the heart with the body-weight in each group When plotted as a graph (Fig 5) these values all fall in the vicinity of a mean curve There is, however, one exception, namely, the group of Marathon runners, whose hearts are unduly large

of the right auricle,[1] while the left border and the apex of the heart are formed by the left ventricle Above the heart lie the great vessels these form the vascular pedicle, on the right is the superior vena cava, in the centre the aorta, and on the left the pulmonary artery Viewed from this aspect the aortic arch is passing almost directly backwards The only part of the left auricle visible from the front is the auricular appendix and, in order to see the body of the left auricle, you have to turn the model round and look at the back of the heart

Fig 7 is an X-ray photograph of a normal heart in a young person In it we can recognize the outline of the different chambers From below upwards, on the right heart border are the right auricle (R A), and above it the superior vena cava (S V C), at the top of the left border is the aortic knuckle (Ao), that is the part of the aorta forming the transverse portion of the arch passing backwards to the left of the spine Below the aortic knuckle is the pulmonary artery (P A) which is continuous with the conus of the right ventricle, and finally we come to the apex of the heart which is formed by the left ventricle (L V) In young subjects the pulmonary conus is more prominent than in older people Immediately below the apex of the heart lies the stomach (St) which in this case is outlined by a bubble of gas in the fundus On the inferior aspect, however, one cannot differentiate between the shadow of the liver lying below the right cupola of the diaphragm and that of the heart above it

By injecting a radio-opaque substance into the veins of the arm and exposing a series of films at intervals of one second Robb (1939) and his colleagues in America were able to outline the different chambers of the heart and the great vessels This method (angio cardiography) is especially helpful in the diagnosis of congenital heart lesions (see p 112)

[1] Anatomists now call the auricle the atrium but with occasional exceptions such as atrial septal defect physicians (and radiologists) still use the old terminology

Fig 7a Teleradiogram of normal heart of a young subject in the Postero anterior (P A) position (*i e with subject facing screen*)

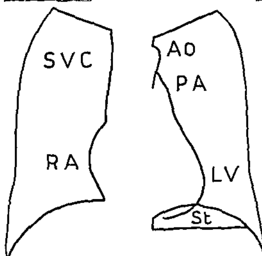


Fig 7*b*

Ao — aortic knuckle
P A — pulmonary artery
L V — left ventricle
S V C = superior vena cava
R A = right auricle
St = stomach

In different diseases the strain falls on different chambers of the heart, these tend to enlarge and so to produce characteristic alterations in the shape of the heart shadow Let me take a simple example In patients with aortic stenosis the left ventricle experiences difficulty in discharging its contents through the narrowed orifice It has to do more work, and, like the muscles of the blacksmith's arm, it hypertrophies This is a true 'work hypertrophy' of the left ventricle and is represented in the radiogram by a leftward extension of the apical portion of the heart shadow A similar but greater enlargement of the left ventricle occurs in patients with aortic incompetence, this renders the cardiac silhouette somewhat boot shaped (Fig 8) with an increased concavity of the left border

Now let us consider the reason for these changes When the aortic valve is incompetent, some of the blood leaks back into the ventricle during diastole and, in consequence, the peripheral circulation is robbed of a part of its blood supply In order to make good the deficiency in the peripheral circulation, the left ventricle has to discharge a larger volume of blood at each beat so that, in spite of some leaking back into the ventricle, there is still sufficient reaching the periphery Whereas in aortic stenosis there is only hypertrophy, in aortic incompetence the increased stroke-volume necessitates dilatation as well as hypertrophy, hence the heart is larger

In the patient from whom Fig 8 was taken the aortic incompetence was due to syphilitic disease of the aorta which had spread to the aortic valves and rendered them incom petent The vascular pedicle is increased in width, this is due partly to dilatation of the aorta and partly to what is known as 'uncoiling' of the aorta, the descending aorta which is not normally visible in this view can be seen lying side by side with the ascending aorta

By rotating the patient through an angle of 45° so that his

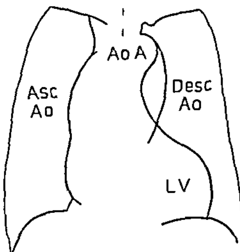


Fig 8*a* Postero anterior (P A) teleradiogram from a case of cardio aortic syphilis showing grossly dilated aorta and enlarged left ventricle

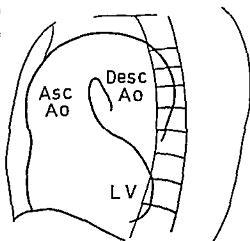
Note great increase in width of the vascular pedicle due to dilatation and uncoiling of the aorta the ascending and descending portions lying side by side instead of being superimposed

Fig 8*b*

Outline drawing of Fig 8*a*

Ao A aort c arch
Desc Ao descending aorta
Asc Ao ascending aorta
L V = left ventricle

FIG 9*a* Left obl que rad ogram f om same case as F g 8 show ng great d latat on of the ascend ng and descend ng aorta



F G 9*b*
Outl ne draw ng of F g 9*a*
Desc Ao descend ng aorta
Asc Ao = ascend ng aorta
L V apex of left ventr cle

left shoulder comes in contact with the X-ray screen, it is possible to visualize the whole course of the thoracic aorta (Fig 9) In this, the *left oblique position*, we see the ascending aorta in front, the arch in the centre, and the descending aorta behind In this patient all three were grossly dilated The apex of the enlarged left ventricle is well seen from this aspect

Now let me consider the changes observed in the shape of the heart when the increased burden falls on the right ventricle, taking as an example mitral stenosis Obstruction at the mitral orifice throws more work on the left auricle which tends to dilate and hypertrophy The increased pressure in the auricle leads to back-pressure on the lungs and, in order to force the blood through the congested lungs, the right ventricle has to hypertrophy Fig 10, which is a post-mortem specimen from a patient who had mitral stenosis, serves to illustrate certain characteristic features In the first place, the left heart border is straight instead of concave, as it was in the case of left ventricular enlargement, and there is a bulge formed by the conus of the right ventricle Roughly speaking, the heart is more rectangular Secondly, two components enter into the formation of the right heart border, of these the left auricle forms the upper and the right auricle the lower arc Fig 11 is an X-ray taken from a patient with mitral stenosis, in it the shape of the cardiac silhouette is very similar to that of the post-mortem specimen

When the left auricle enlarges, it extends backwards towards the spine, displacing the oesophagus which lies in direct contact with the posterior surface of the heart, and can be visualized by giving the patient a mouthful of barium to swallow The most favourable position for studying the left auricle is the *right oblique*, in which the patient is rotated through an angle of about 45° so that his right shoulder comes in contact with the screen In this position, in normal subjects,

the posterior mediastinum appears as a clear area bounded in front by the posterior surface of the heart, and behind by the spine, but enlargement of the left auricle produces a



FIG 10 Photograph of autopsy specimen of heart from

sickle shaped impression on the oesophagus which is displaced backwards (Fig 12) Such backward enlargement is limited by the spine, and when the auricle continues to dilate it pushes its way rightwards and makes its appearance on the right heart border

It may seem curious that enlargement of the left auricle should appear on the right heart border and enlargement of the right ventricle on the left border, but as the heart lies in the body, these two chambers are situated not to the left and right but at the back and front of the heart

left shoulder comes in contact with the X-ray screen, it is possible to visualize the whole course of the thoracic aorta (Fig 9) In this, the *left oblique position*, we see the ascending aorta in front, the arch in the centre, and the descending aorta behind In this patient all three were grossly dilated The apex of the enlarged left ventricle is well seen from this aspect

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FIG 12*a* Right oblique radiogram from same case as Fig 11 showing backward enlargement of left auricle

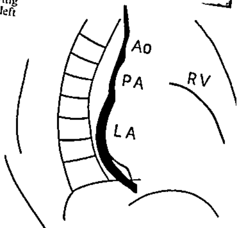


FIG 12*b* Outline drawing of Fig 12*a* In this and the following diagrams the barium filled oesophagus is represented in black
Ao aortic impression
PA pulmonary artery impression,
LA left auricular impression
RV = conus of right ventricle

Fig 13*a* Right oblique radiogram from same case as Figs 8 and 9

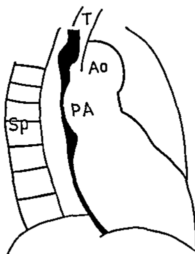


Fig 13*b* Outline drawing of Fig 13*a*
P A = pulmonary artery impression on oesophagus
Sp = spine T = trachea
Ao = aorta

With Fig 12, compare Fig 13 which was taken from the patient with aortic incompetence to whom I have previously referred In this case there was no enlargement of the left auricle, and consequently the oesophagus is not deflected backwards

When the right ventricle is greatly enlarged it may form part of the right heart border This is easily recognized on screening, since systole of the right ventricle is synchronous with that of the left Similarly, great enlargement of the left auricle may produce a prominence on the left heart border and will then be seen to pulsate asynchronously with the left ventricle

In certain types of congenital heart disease such as Fallot's Tetralogy the right ventricle is enlarged, but the shape of the cardiac silhouette is very different from the somewhat rectangular heart shadow seen in cases of mitral stenosis (Fig 11) In the P–A view (Fig 14) the heart is boot-shaped with a concave instead of a straight or convex left border It is not unlike the enlarged left ventricle in cases of aortic incompetence (Fig 8) In the left oblique position, however, the enlarged right ventricle can be seen bulging forwards towards the sternum, and the apex of the heart does not extend backwards across the spine Roesler (1943) suggests that the explanation of the difference in shape of the enlarged right ventricle in Fallot's Tetralogy from that in mitral stenosis is that the direction of the outflow tract of the ventricle is less vertical in the former than in the latter, since most of the blood is entering the aorta instead of the pulmonary artery

There are two special modifications of radiographic technique which are occasionally used in examination of the heart—'Kymography' and 'Tomography'

In *Kymography* an opaque grid with a series of horizontal slits is placed between the heart and the film By keeping the grid stationary and moving the film, a jagged outline of the

heart shadow (Fig 15) is obtained, each tooth on this jagged edge being a record of the movement of a particular segment of the heart surface during systole and diastole Thus excessive or deficient pulsation can be readily detected

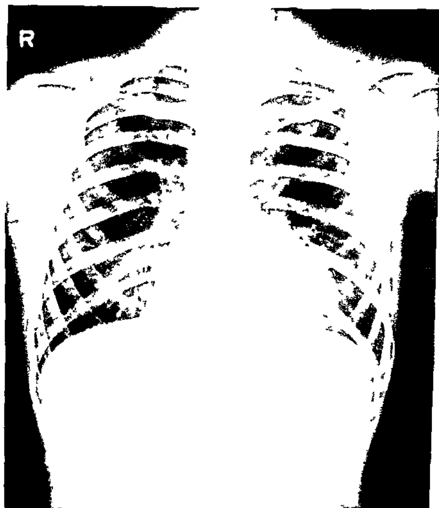


Fig 14 P–A radiogram from a case of Fallot s Tetralogy (see p 110) showing 'boot shaped heart with concave left border, and apex formed by right ventricle

The principle involved is very simple, and is illustrated diagrammatically in Fig 16 *AD* is a slit in the grid, the shadow of the heart border moves from *C* to *B* during systole and is recorded as a sphygmogram on the film, which during the

exposure moves from X to Y. The distance XY is less than the distance between two adjoining slits, so that each slit

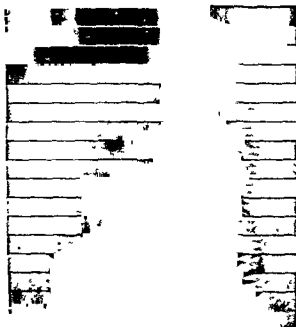


FIG 15. Kymogram, in left oblique position, from a case of cardio-aortic syphilis, showing wide and tortuous aorta
(*Kindly lent by Department of Roentgenology, Johns Hopkins Hospital*)

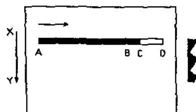


FIG 16 Diagram to illustrate principle of kymography

records the movement of a separate segment of the heart surface In the kymogram (Fig. 15), each of the transverse bands corresponds to the movement of one slit Thus the

movement of the grid as a whole yields a series of records, three heart cycles being recorded in each slit Great hopes were entertained that this method might throw new light on clinical problems These hopes, however, have not been realized, owing to certain fallacies which the method entails The size of the indentations in the kymogram depends not only on the local movement of the heart wall, but also on

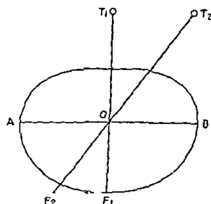


FIG 17 Diagram to illustrate the principle of tomography

the movement of the heart as a whole, while only those movements of the heart wall which happen to be in the plane of the grid are accurately recorded

Tomography helps us to differentiate between the shadows of superimposed organs If the X-ray tube and the film be made to move in opposite directions parallel to one another, but at different rates, the only objects which remain in focus are those in the plane of the axis of a lever controlling the respective movements of tube and film For example, if the tube (Fig 17) be moved from T_1 to T_2 while the film is kept stationary, the image of an object at O would be a blurred image F_1–F_2, but if the film be made to move in unison with the tube through a distance F_1 to F_2 the image of all structures in the plane AB will remain in focus while the images of objects situated in planes closer to or farther from the tube

than *AB* will appear blurred By altering the relative lengths of the two limbs (*TO* and *FO*) of the lever, it is possible to bring into focus objects in any desired plane of the body while eliminating to a considerable extent the shadows of objects situated in other planes Tomography has proved more useful in examination of the lungs than of the heart, but it has certain limited applications to cardiology which may be further developed in the future Calcification of the heart valves, for example, can be demonstrated much more clearly in a tomogram than in a 'straight' film

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CHAPTER III

VALVULAR LESIONS

In considering the sequence of events which constitute the cardiac cycle and the manner in which they may be modified by disease of the heart valves, for the sake of simplicity I shall confine my attention to the left side of the heart which clinically is the more important, for, owing to the fact that the pressure is higher in the systemic than in the pulmonary circuit, the mitral and aortic valves are subject to greater wear and tear than the tricuspid and pulmonary and so are more liable to be damaged by infection

INTRAVENTRICULAR PRESSURE

In health ventricular systole follows immediately on auricular systole, in fact the two actually overlap, but, as soon

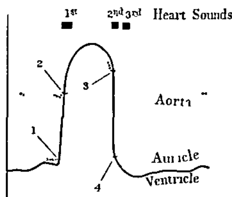


FIG 18 Intracardiac and aortic pressure curves (Modified from Wiggers) (1) mitral valve closes (2) aortic valve opens (3) aortic valve closes (4) mitral valve opens

as the pressure in the ventricle rises above that in the auricle, the mitral valve closes (Fig 18) This is succeeded by isometric contraction of the ventricle, then the aortic valve opens and the pressure curves in ventricle and aorta follow one another closely, at first rising, later falling With the cessation of

systole the pressure in the ventricle drops suddenly, the aortic valve closes, and about 0 1 seconds later the mitral valve opens and the blood which had been accumulating in the auricle throughout systole rushes into the empty ventricle

HEART-SOUNDS

Clinically, these different phases of the cardiac cycle are recognized by their relation to the heart-sounds

Two heart-sounds are audible in all healthy people Expressed in the simplest terms, the first heart-sound is produced by contraction of the ventricles and the second by closure of the semi-lunar valves Thus the first sound marks the beginning and the second sound the end of the ventricular systole How are we to tell which is the first and which the second? The second sound is generally sharper and higher pitched than the first, but the character of the sounds is not always a reliable guide. When the heart is beating slowly we can tell by the rhythm, for the first sound is followed by a short pause and the second by a long pause but when the heart accelerates diastole is curtailed to a greater extent than systole (see Fig 27) and the two pauses may be of equal length Clinically the surest way of timing the sounds is by the carotid pulse which occurs 0 1 seconds after the commencement of ventricular systole and so is almost synchronous with the first heart-sound In graphic records (Fig 19) the first heart-sound is synchronous with the *R* wave of the electrocardiogram and the second sound with the *T* wave [1]

TRIPLE RHYTHM

The term 'triple rhythm' means that three instead of two sounds accompany each cardiac cycle When the additional

[1] This is only approximately true since the electrical and mechanical changes in the heart are not absolutely synchronous For research purposes a tracing of the venous pulse or of the apical impulse is more reliable and has the additional advantage of indicating the time relations of events occurring during diastole

sound occurs early in diastole we call it the 'third heart-sound', but when it occurs during auricular systole we call it the 'fourth heart-sound' (Fig 19)

The third heart-sound (or 'reduplicated second sound') may be physiological and is probably produced by the sudden

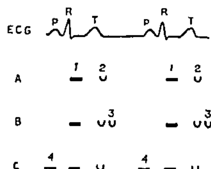
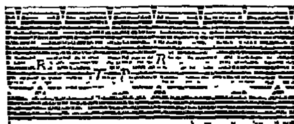
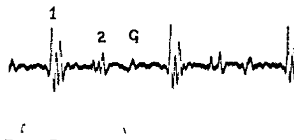


Fig 19 Diagrammatic representation of heart sounds to illustrate time relation of third heart sound (3) and fourth heart sound (4) to first (1) and second (2) normal heart sounds
E C G — Electrocardiogram
A — Normal heart sounds
B — Protodiastolic gallop
C = Presystolic gallop

rush of blood into the ventricle when the mitral valve first opens It is heard in many perfectly healthy young people, but it may also be pathological, an additional sound in early diastole known as the 'opening snap' of the mitral valve being heard in patients with mitral stenosis (see Fig 25) The fourth heart-sound (or 'reduplicated first sound') (Fig 20) is always pathological and may be a sign of very grave significance it is 'the cry of the heart for help' It warns us that the last reserves have been called up and that the heart is struggling against desperate odds In a consecutive series of sixty-two of my patients in whom this sign was present, only fifteen lived for more than eighteen months (Bramwell, 1936) The presence of a fourth heart-sound generally signifies failure of the left ventricle, which is due in some cases to inability to bear the additional burden imposed upon it by high blood-

pressure, or by a lesion of the aortic valve, and in others to grave myocardial damage produced either by an acute infection such as diphtheria or by infarction of the ventricle following coronary occlusion The exact mechanism of production of the fourth heart-sound is still a matter of dispute,





the *R* *T* and *P* waves of the electrocardiogram

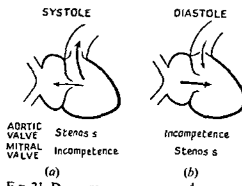
but there is considerable evidence in support of the hypo thesis that it is produced by vibrations of the atonic ventricular wall which is set in motion by the sudden filling of the ventricles during auricular systole (Bramwell 1935) The terms 'Gallop' and Canter' rhythm have been applied to triple rhythm on account of its resemblance to the cadence of the sounds produced by a horse galloping or cantering

HEART-MURMURS

Disease may damage a valve cusp rendering the valve incompetent and so permitting the backward regurgitation of

blood, or it may produce narrowing (stenosis) of the valvular orifice and so obstruct the forward passage of the blood Both defects set up eddies in the blood-stream which give rise to murmurs

During systole of the ventricle (Fig 21*a*), the mitral valve is closed and blood is flowing into the aorta Hence the valvular lesions which give rise to systolic murmurs are those produced



arrows indicate valvular incompetence

by incompetence of the mitral valve or by obstruction at the aortic orifice Conversely diastolic murmurs (Fig 21*b*) are due to aortic incompetence or mitral stenosis Mitral murmurs are distinguished from those produced at the aortic orifice by the fact that they are most clearly heard in the vicinity of the cardiac apex whereas aortic murmurs are best heard either at the base of the heart or along the left border of the sternum

Systolic murmurs replace or follow the first heart sound and so are synchronous with the carotid pulse, while in the case of diastolic murmurs there is an appreciable interval between the pulse and the murmur It may, however, be very difficult to time a murmur with certainty when the heart is beating rapidly, and I am inclined to think that we lay too much emphasis on the timing of murmurs and too little on

their character The typical short, rough, crescendo, presystolic murmur of mitral stenosis which ends in a loud banging first heart-sound, and is often preceded by a low-pitched irregular rumble, is quite unlike the soft blowing apical murmur which dies away gradually and smoothly in a much more orderly fashion The latter is always systolic Likewise the 'machinery murmur' of the patent ductus (see p 110), one of the commonest congenital lesions, could hardly be mistaken for anything else Or again, the faint, soft, high-pitched whiff to the left of the sternum which one hears in many cases of aortic incompetence is so typical that there is never any doubt that it is diastolic The experienced clinician recognizes these murmurs by their character He does not have to time them

A damaged valve cannot be repaired, but a systolic murmur does not necessarily signify a damaged valve, systolic murmurs are heard in patients with fever, anaemia, thyrotoxicosis, and other conditions, but when the underlying disease is cured these murmurs generally disappear

It may be very difficult to decide whether a systolic murmur is or is not due to organic disease This problem frequently arose during the war in the examination of recruits for the Services Even amongst cardiologists of repute there exists a difference of opinion regarding the significance of systolic murmurs, though most agree that loud murmurs are generally organic, but, so long as such a difference of opinion does exist, the patient should always be given the benefit of the doubt, and his activities should never be restricted on account of a systolic murmur, unless there is other evidence of heart disease

The last word on systolic murmurs has not yet been spoken Here is a fruitful field for clinical research by correlating the murmurs we hear with the stethoscope, and which we can now record by graphic methods, with the actual condition of

the valves found at autopsy, and with alterations in the size and shape of the different chambers of the heart as seen by X-rays during life

MITRAL STENOSIS

The cusps of the mitral valve spring from a continuous curtain, consequently the contraction which follows healing of inflammation in this valve draws the cusps together and leads to narrowing of the orifice—'mitral stenosis'

In the production of all obstructive murmurs two factors are concerned—the size of the orifice and the rate at which the blood is flowing through it, the one is static, the other dynamic No matter how small the orifice, unless the blood-flow attains a certain critical velocity, no murmur is produced There is an inverse relation between the degree of stenosis and the velocity of the blood-flow required to produce an obstructive murmur Anatomically, the degree of stenosis is measured by the size of the orifice, *physiologically* by the amount of blood it can transmit in unit time

Fig 22 illustrates the importance of the dynamic factor Since this patient had partial heart block (see p 89) the relation of auricular to ventricular systole varied from cycle to cycle, and in some cycles the auricle contracted alone without the ventricle The lower tracing is a record of the heart-sounds, and the upper one a synchronous electrocardiogram Below I have represented diagrammatically the sequence of auricular and ventricular systole in the different cycles *In the first cycle* (P_1) *ventricular systole* follows immediately on auricular systole In the second cycle (P_2) there is a longer interval between them, and in the third cycle (P_3) the auricle contracts alone, there is no ventricular response This sequence is then repeated, the fourth cycle (P_4) being similar to the *first* (P_1)

In the first cycle (P_1) there is a loud first heart-sound but

no murmur in the second cycle (P_2) there is a murmur preceding the first heart sound in the third cycle (P_3), in which the auricle contracts without the ventricle, there is a loud murmur but no first heart-sound The fourth cycle (P_4) is

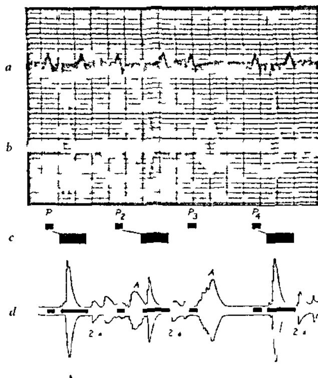


FIG 22 Phonocardiogram with synchronous electrocardiogram from a case of mitral stenosis with partial heart block (a) Electrocardiogram (b) Phonocardiogram (c) Diagram showing relation of auricular to ventricular systole (d) Outline drawing of phonocardiogram heavy lines show time relation of auricular and ventricular systole to heart sounds and murmurs

similar to the first (P_1) We were extremely puzzled when we first listened to this patient's heart Why did the sounds vary in this extraordinary way from cycle to cycle, and why, if the patient had mitral stenosis, was there no murmur in some cycles? Without a graphic record it would have been difficult to solve this problem The absence of the murmur in the first

(P_1) and the last (P_4) cycles is explained by the fact that both these followed a missed beat and, during the prolonged diastole preceding these cycles, the auricle contracted twice When it contracted the second time, it was unable to force sufficient blood through the mitral orifice to produce a murmur, for the ventricle was already full During the intermission, on the other hand, the auricle contracted (P_3) early in diastole and discharged its contents with considerable velocity into the empty ventricle, producing a loud murmur. The variability of the murmur, in this case, depended entirely on the intraventricular pressure at the time the auricle contracted

In health the rate of blood-flow through the mitral orifice varies greatly during the different phases of diastole This can be seen from the curve of ventricular filling (Fig 23*a*), in which the volume of blood in the ventricle is plotted against time When the mitral valve first opens, the pressure in the auricle is much higher than in the ventricle, and the blood which had accumulated in the auricle during ventricular systole rushes in a swirling torrent into the empty ventricle A distinguished surgeon once told me that, in all his experience, nothing had left a more vivid impression on his mind than the sensation of the blood rushing past his finger when he introduced it into the heart to operate on the mitral valve During mid-diastole, auricle and ventricle behave as a single chamber, there is little difference in pressure on the two sides of the mitral orifice, and the rushing torrent of early diastole is converted into a quietly flowing stream Finally, when the auricle contracts, the rate of blood-flow is again increased Thus blood-flow from auricle to ventricle is rapid during early diastole and pre-systole, but much less rapid during mid-diastole

Severe stenosis limits the amount of blood which can pass through the mitral orifice in early diastole, the rate of filling

of the ventricle then becomes more uniform (Fig 23*b*), and the pressure in the auricle remains much higher than that in the ventricle throughout diastole, with the result that even during mid diastole, the velocity of the blood-current through the mitral orifice is considerable and an obstructive murmur is heard throughout diastole When, however, there is only

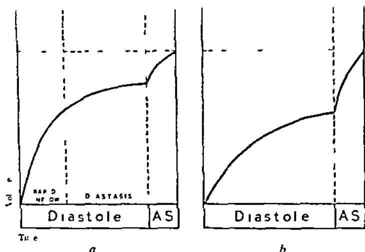


Fig 23 Ventricular filling curve (*a*) normal (*b*) mitral stenosis

a moderate degree of stenosis the orifice is still adequate to transmit the slowly flowing stream during mid diastole Consequently this phase is silent, and it is only when the rate of blood-flow is increased during early diastole or auricular systole that a murmur is produced When there is only slight stenosis the mitral orifice may be large enough to cope with the rapid blood-flow in all phases of diastole when the subject is at rest, but, if the rate of blood-flow be increased by exercise, a murmur is heard Hence, by auscultation, it is possible not merely to diagnose mitral stenosis, but to form a rough estimate of the degree of stenosis present When stenosis is slight, no murmur may be heard at rest, but a presystolic (Fig 24) or early diastolic murmur can be elicited by exercise, in moderate stenosis there is a murmur at rest, but it is

confined to presystole or early diastole, mid-diastole being silent, whereas in severe stenosis a long rumbling murmur fills the whole of diastole (Fig. 25).

One can carry this hypothesis a stage farther. If the production of an obstructive murmur depends on the size of the orifice relative to the rate at which the blood is flowing

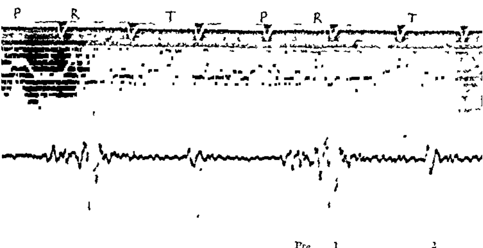


FIG 24 Phonocardiogram recorded with Matthews's oscillograph and condenser microphone, from a patient with slight mitral stenosis Pre – presystolic murmur 1 – first heart sound 2 – second heart-sound

through it, theoretically a murmur might be produced either by a narrowed orifice or by an increased rate of blood-flow When the cardiac output is increased by exercise, or when the heart is overactive, as in hyperthyroidism, the first heart-sound may be roughened and accentuated, and then bears a close similarity to that of mitral stenosis (Bramwell, 1943) This hypothesis is supported by *Paul Wood's (1950)* observations on the apical diastolic murmur heard in patients with a patent ductus. This murmur is due to an increased blood-flow through the mitral orifice and is indistinguishable from that of mitral stenosis, but disappears when the ductus is ligated.

This is not merely an academic matter, but a problem of

considerable clinical importance, for it is often very difficult to be certain whether a roughened and accentuated first heart-sound is due to a slight degree of mitral stenosis or merely to an overacting heart I believe the reason for this difficulty is that there is no sharp dividing line between physiological

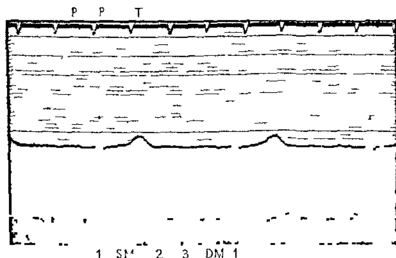


Fig 25 Electrocardiogram and phonocardiogram recorded with a Matthews's oscillograph and condenser microphone from a patient with advanced mitral stenosis (1) first heart sound (S M) systolic murmur (2) second heart sound (3) third heart sound (D M) diastolic murmur

and pathological stenosis, that is to say between cases in which the increased rate of blood flow is too great for the normal orifice, and those in which the narrowed orifice is too small to transmit the normal blood flow

In Table IV I have summarized the physical signs in the various grades of mitral stenosis When the orifice is normal and the blood flow is normal the heart-sounds are normal An accentuated and roughened first heart sound may be due either to a slight degree of stenosis or merely to an overacting heart A murmur confined to pre-systole or early diastole, which can only be elicited by exercise, is suggestive of a slight degree of stenosis, but, if present at rest it suggests a moderate

stenosis A full diastolic murmur heard at rest suggests severe stenosis

TABLE IV

Grades of Mitral Stenosis

Orifice	*Blood flow*	*Heart sounds*
1 Normal	normal	Normal
	increased	Accentuated first heart sound
2 Slight stenosis	normal	
	increased	Murmur in pre systole or early* diastole
3 Moderate stenosis	normal	
	increased	Murmur throughout diastole
4 Severe stenosis	normal	

* Some cardiologists call this the mid diastolic murmur since it follows the third and not the second heart sound

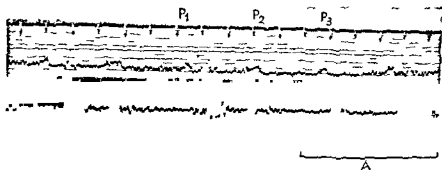
This conception of the various grades of mitral stenosis is a purely theoretical conception it is merely as a working hypothesis *Parts of this hypothesis will, I believe, stand the* test of time, for instance, I think there can be no doubt that we are right in saying that the patient whose murmur can only be elicited by exercise has a lesser degree of stenosis than one in whom the murmur is present at rest Other parts of this hypothesis may have to be modified when new facts are brought to light

When the auscultatory signs are equivocal, one has to look for other evidence to establish the diagnosis of mitral stenosis In this lesion the strain falls first on the left auricle, which tends to dilate and hypertrophy This is easily recognized by radiography (see Fig 12, p 29) Later, back pressure on the lungs raises the pressure in the pulmonary circuit and imposes a strain on the right ventricle, dilatation of which gives rise to a *characteristic alteration in the shape of the*

heart, with prominence of the pulmonary conus (see Fig 11, p 28) Congestion in the pulmonary circuit is responsible for dyspnoea, the characteristic symptom of mitral stenosis, and not infrequently leads to haemoptysis from rupture of the engorged lung capillaries

The auriculo systolic murmurs of mitral and tricuspid stenosis differ from all other heart-murmurs in that they are crescendo in character The reason for this peculiarity has given rise to much speculation I believe it to be that the ventricle begins to contract before the auricle relaxes so that the terminal vibrations of the auricular murmur overlap and are reinforced by the initial ventricular vibrations of the first heart-sound (Fig 26)

Ventricular filling normally depends, in part, on auricular systole and, in part, on the passive inflow from the great veins during mid-diastole under some conditions the former, under others the latter, is the more important When the heart accelerates, diastole is curtailed to a much greater extent than systole, as shown in Fig 27 This record was taken from a normal subject—after an inhalation of amyl nitrite, a drug which, amongst other effects, produces cardiac acceleration Eighty seconds after the inhalation, when the heart-rate was 130 per minute, the whole diastolic portion of the cardiac cycle was occupied by auricular systole, which followed immediately on ventricular systole As the heart decelerated, diastole became longer, as shown by the increasing interval between the *T* wave of one cycle and the *P* wave of the next In these three records it is 0, 0 1, and 0 3 second respectively The more rapid the heart-rate the greater is the contribution made to ventricular filling by auricular systole In the first record, not only does auricular systole occupy the whole of diastole, but the amount of blood discharged by the auricle is increased, since the intra-auricular volume is greater at the beginning than at the end of diastole, when the auricle



a

P3
1 sound

b

Fig 26 *a* Synchronous electrocardiogram (uncalibrated) and phonocardiogram (recorded with Matthews's oscillograph and condenser microphone) from a patient with Graves's disease and varying 2 : 1, 3 : 1 A–V block Time intervals 0 2 sec

... Fig 26*a* In the first cycle of this record (P_3)
... auricular
...ions of the
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normally contracts Under these circumstances the additional impetus of auricular systole is superimposed on that of the rapid inflow, which normally occurs during early diastole, yielding a summation of the two effects



FIG 27 Electrocardiograms and optical carotid sphygmo

after commencement of inhalation Heart rate 100 *T–P* interval 0 1 sec (*c*) 360 seconds after commencement of inhalation Heart rate 73 *T P* interval 0 3 sec

AORTIC REGURGITATION

In contrast to the mitral the cusps of the aortic valve are discrete, consequently, when contraction occurs during healing of the inflamed valve, the cusps tend to be drawn apart, with the result that the valve becomes incompetent and blood regurgitates into the ventricle during diastole, producing a characteristic murmur

Let us now consider the consequences of aortic regurgitation Since the blood is able to escape in two directions, back into the ventricle as well as to the periphery, the peripheral circulation is robbed of part of its blood and, since the organ most sensitive to a deficient blood supply is the brain, patients with aortic regurgitation manifest symptoms of cerebral anaemia, such as faintness and dizziness To maintain an adequate circulation, under these circumstances, the left ventricle has to increase its output per beat, this entails both

dilatation and hypertrophy, and great enlargement of that chamber may occur (see Fig 8, p 24)

In aortic regurgitation the combination of an increased stroke-volume of the left ventricle and a low diastolic pressure give to the pulse a peculiar 'water-hammer' character to which I shall refer later (p 58)

There is another interesting difference between mitral stenosis and aortic incompetence If one takes 100 cases of mitral stenosis and represents graphically the age at which they are diagnosed clinically, one finds that they conform to what statisticians call a 'normal frequency curve' If we do the same for cases of aortic incompetence, we find that the curve has two humps instead of one This is due to the fact that, whereas mitral stenosis is always rheumatic, aortic incompetence may be due either to rheumatism or to syphilis, it has a dual aetiology and, while it is probable that aortic incompetence in a patient of 25 is rheumatic, aortic incompetence developing at the age of 40, in a previously healthy subject, is almost always syphilitic

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CHAPTER IV

THE PULSE

WITH each contraction of the heart, a wave of increased pressure passes along the arteries, this we call the pulse It is important to distinguish between this wave of pressure which is transmitted from heart to periphery and the actual movement of the blood-stream The pulse-wave is like a ripple travelling over the surface of moving water

The pulse has interested physicians since the dawn of medicine, when it was believed to be due to the rhythmic efforts of the vital spirits to escape from imprisonment! Graphic registration by means of the sphygmograph paved the way to further advances in our knowledge of the subject Dudgeon (1892), in the little book describing his sphygmograph, wrote as follows

> The physician of old made his diagnosis chiefly by observation of the pulse and tongue But, as the tongue could be rapidly inspected, and anyone could judge of its foulness or cleanness as well himself, he concentrated his attention mainly on the pulse, in the feeling of which there was always scope for affecting the possession of peculiar skill and insight To the uninitiated, who regarded the doctor as the depository of occult knowledge, and received his dicta as though they were oracles, there was something very imposing in his method of pulse palpation The fingers of the right hand daintily grasping the patient's wrist, while the doctor's eyes were riveted on the loud ticking gold chronometer he held in his left hand his head gravely nodding the while synchronously with the arterial pulsations—all this formed a picture calculated to inspire beholders with reverence and awe

The modern patient has, I fear, lost some of this reverence and awe—he is too well informed—but nevertheless he still expects you to feel his pulse!

Palpation of the pulse tells us its rate, rhythm, and charac-

ter, and enables us to judge of the condition of the arterial wall

PULSE-RATE

The pulse-rate in the majority of healthy adults is in the neighbourhood of 70 per minute

A slow heart-rate is an advantage for *bradycardia* makes it possible to double or even to treble the cardiac output without an excessive increase in heart-rate, and athletes, who indulge in those forms of sport involving severe and prolonged exertion, usually have slow pulses the same is true of the animal kingdom (Clark, 1927) For example, the resting pulse-rate of the hare, an exceedingly athletic animal, is under 70, while that of its cousin, the rabbit, a sedentary creature, who never strays far from his burrow, is over 200 The difference in rate, in this case, is due to the influence of the vagus, for, after section of this nerve, the rabbit's heart-rate was found to rise to 321 and that of the hare to 264 To compensate for the difference in pulse-rate, the hare's heart, relative to its body-weight, is three times as large as the rabbit's In a previous chapter (p 19) I referred to a similar relation between the size of the heart and the rate of the pulse in healthy young men

At birth the pulse-rate is about 120 per minute, throughout childhood and adolescence it falls, but does not reach its lowest value until the age of 25

Tachycardia may be due to various causes, of which the most common is nervousness Many people are scared at the very thought of a medical examination and, during the war, I was often asked by Medical Boards to decide whether persistent tachycardia in a recruit was physiological or pathological

In febrile diseases the increased heart-rate is proportional to the increased metabolism There are, however, certain

notable exceptions, such as the enteric group of infections, in which the pulse-temperature ratio is upset, the pulse being unduly slow as compared with the temperature, a helpful sign in the diagnosis of these diseases

Various tests of cardiac efficiency are based on the response of the pulse to exercise The test generally employed, in the examination of recruits for the Services, is to take the resting pulse-rate and then ask the subject to place one foot on a stool, about 18 inches in height, and raise the body 20 times in 60 seconds The pulse-rate is taken immediately after the cessation of exercise and again one minute after the exercise, by which time it should have returned to normal The most significant figure in all such exercise-tolerance tests is the *time taken by the heart to return to its resting rate after* the cessation of exercise It is also important to note whether the exercise provokes breathlessness or other signs of distress

PULSE FORM

Wiggers (1923) has shown experimentally in dogs that the pressure changes in the large arteries near the heart correspond closely to those taking place within the left ventricle, but, in the course of transmission from heart to periphery, the pulse-wave is subject to the damping influence of the elastic arterial walls This modifies the form of the sphygmogram by rounding off the rapid deflexions, as shown in Fig 28, in which the upper record is taken from the carotid and the lower from the radial artery of a normal subject In both records three waves can be recognized The first, or percussion wave (*P*), is due to the arrival of the pulse in the artery, the second or tidal wave (*T*), is probably reflected from the periphery, and the third, or dicrotic wave (*D*), is produced by the rebound of the blood forced back against the closed semilunar valves by the elastic recoil of the aorta in early diastole

In most patients only the percussion wave can be felt on palpation of the pulse, but occasionally two waves can be detected, the second being either the tidal or the dicrotic The tidal wave is palpable in some cases of aortic incompetence combined with stenosis (Fig 29*a*), the dicrotic wave



FIG 28 Sphygmograms (optically recorded) from normal subject Car Carotid Rad radial *P* percussion wave *T* tidal wave *D* — dicrotic wave



FIG 29 (*A*) Pulsus bisferiens (*B*) Dicrotic pulse *P* percussion wave *T* — tidal wave *D* dicrotic wave

in conditions such as typhoid fever, in which ventricular systole is poorly sustained (Fig 29*b*) The former is known as the 'pulsus bisferiens , the latter as the 'dicrotic pulse' When two waves are palpable, two sounds instead of one are heard accompanying each heart-beat, while the armlet is being deflated in taking the blood pressure (Fig 30)

The character of the pulse depends on the pressure and the rate of change of pressure within the artery The most striking deviations from the normal type of sphygmogram are seen in patients *with lesions of the aortic valve* (Fig 31) Aortic stenosis limits the ventricular output during the initial

stage of systole, consequently the pressure in the aorta rises more slowly than in health and the systolic pressure is low In this condition, since the ejection phase of systole is

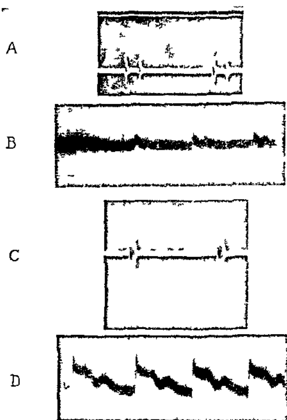


FIG 30 Phonoarteriograms and sphygmograms from pati
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prolonged, the apex of the sphygmogram is formed by the tidal and not by the percussion wave Conversely, when the aortic valve is incompetent, the hypertrophied and dilated left ventricle discharges an abnormally large volume of blood into the aorta early in systole and consequently the systolic

In most patients only the percussion wave can be felt on palpation of the pulse, but occasionally two waves can be detected, the second being either the tidal or the dicrotic The tidal wave is palpable in some cases of aortic incompetence combined with stenosis (Fig 29a) the dicrotic wave



Fig 28 Sphygmograms (optically recorded) from normal subject Car Carotid Rad radial *P* percussion wave *T* tidal wave *D* dicrotic wave

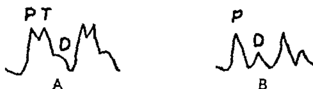


Fig 29 (A) Pulsus bisferiens (B) Dicrotic pulse
P percussion wave *T* – tidal wave *D* dicrotic wave

in conditions such as typhoid fever, in which ventricular systole is poorly sustained (Fig 29*b*) The former is known as the 'pulsus bisferiens', the latter as the 'dicrotic pulse' When two waves are palpable, two sounds instead of one are heard accompanying each heart beat, while the armlet is being deflated in taking the blood pressure (Fig 30)

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The sphygmogram (Fig 32) is a record of the pressure changes in the radial artery at a particular point it is a 'pressure-time' curve We never think of waves on the sea-shore in that way we think of them as we see them in a painting or in a photograph (Fig 33), where they are repre-

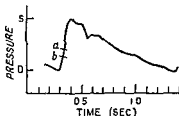


FIG 32 Normal carotid sphygmogram



FIG 33 Waves breaking on the sea shore
(*After woodcut by Oscar Drage*)

sented as a pressure-space relation at a particular instant of time In the photograph we have eliminated time, it is instantaneous, in the sphygmogram we have eliminated space, it is recorded at a particular point, to represent all three attributes simultaneously would require a cinematograph

As a wave advances on the beach, the wave front becomes steeper and steeper and eventually unstable, it loses its balance and crashes over with the formation of breakers The pulse-wave undergoes similar changes as it travels from heart to periphery and, under certain conditions, it also may become unstable, but in a closed system, such as an artery, material breakers cannot develop, for the artery is full of fluid and there is nowhere for the wave to break Under

these circumstances the pulse-wave regains its stability by transferring part of its energy to new waves which are shot out in front (Fig 34) and behind it (Bramwell, 1925). These waves are analogous in terms of mechanical pressure to the material breakers on the sea-shore. They produce a turbulent

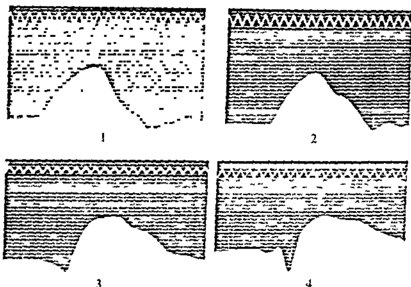


FIG 34 Shadow sphygmograms of waves recorded at different points along the course of a tube filled with mercury Tuning fork marking 100 double vibrations

condition in the fluid in the artery which manifests itself by rapid oscillations of pressure, that can be felt as a thrill

On purely mechanical grounds it can be shown (Bramwell and Hill, 1923) that the conditions favourable to the development of this phenomenon are a low diastolic pressure and a steep pressure gradient along the front of the pulse-wave—the very conditions which are present in patients with free aortic regurgitation (Fig 35)

The 'water-hammer' pulse is not peculiar to aortic regurgitation. It also occurs in other conditions such as arteriovenous aneurysm and patency of the ductus arteriosus, in

which the blood is able to escape in two directions, part going to the periphery and part passing back through the abnormal channel, while in some cases of thyrotoxicosis or profound anaemia the bounding pulse closely simulates that of aortic incompetence

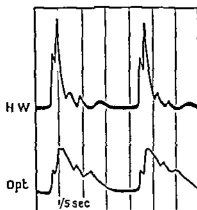


FIG 35 Simultaneous sphygmograms of carotid pulse from a patient with aortic incompetence showing vibrations on the upstroke H W = hot wire sphygmogram Opt = optical sphygmogram

Pulse rhythm will be considered in the next chapter, and we may now consider the problem of pulse tension

BLOOD-PRESSURE

The Arterial Sounds By compressing the arm with a pneumatic armlet we can reduce the tension of the arterial wall This slows the rate at which the pulse-wave travels and renders the conditions favourable for the formation of 'breakers' It is these sudden variations of pressure within the artery that set the arterial wall into rapid vibration and so produce the sharp cracking sounds on which we rely for estimating the diastolic blood pressure in man

Fig 36 is a graphic record of the arterial sounds, taken during deflation of the armlet As decompression of the arm proceeds, the arterial sounds at first become louder and

sharper then, suddenly, they diminish in intensity and change in quality, instead of being clear and sharp they become dull and muffled This abrupt diminution in inten-

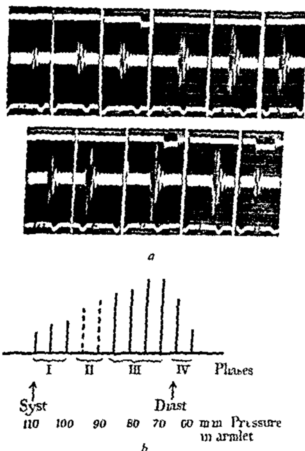


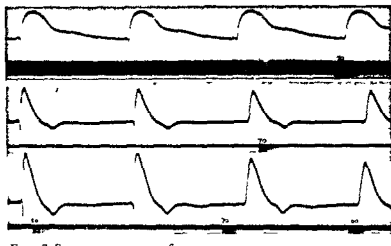
FIG 36 (a) Arterial sounds recorded with electrical stethoscope and string galvanometer Numerals below record indicate pressure in armlet (b) Diagram showing amplitude of excursion in records in 36a

sity and change in quality marks the diastolic end-point and with a further fall in pressure of only a few millimetres, the sounds attain the lower limit of audibility and throughout the remainder of decompression no sound is heard

Some life-insurance companies direct their examiners to take the disappearance of the sounds as the diastolic end-

point They do so because it is more easily recognized than the transition from loud to faint sounds

If, instead of listening with a stethoscope, you palpate the brachial artery immediately below the armlet, you will notice



Note negative pre anacrotic wave in first two cycles in each record

that during the range of decompression when the arterial sounds are loud and sharp the pulse assumes a 'water-hammer' character, but immediately the pressure in the armlet drops below diastolic the 'water-hammer' character is lost

Fig 37 is a graphic record of the pulse in the brachial artery taken during deflation of the armlet over a range of pressures immediately above and below diastolic Above diastolic a rapid negative wave precedes the upstroke of the sphygmogram, but below diastolic this pre-anacrotic wave is absent This negative pre-anacrotic wave is one of the 'breakers' to which I have referred It is responsible for producing the rapid oscillations of the arterial wall which give rise to the sharp cracking sounds

In patients with free aortic regurgitation, it may be difficult to estimate the diastolic blood-pressure, for, whereas in the normal subject the 'water-hammer' character of the pulse is lost when the compressing pressure falls below diastolic, in the patient with aortic regurgitation it persists, and loud arterial sounds are heard throughout the whole of decompression Careful auscultation, however, reveals that a change

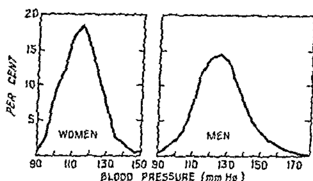


FIG 38 Curves showing range of variation in systolic blood pressure in normal subjects (After Alvarez)

in the *character* of the sounds does occur during the later stage of decompression, and this indicates the diastolic end-point

In patients with aortic stenosis it is often impossible to estimate the diastolic pressure, because the reduced rate of blood-flow through the narrowed orifice renders the pressure gradient along the front of the pulse-wave so gradual that no breakers are formed and consequently no sharp sounds are produced

Normal blood-pressure In health the basal blood-pressure varies considerably in different individuals In a series of university students aged 16–40 (Fig 38) Alvarez (1920) found that the mean systolic pressure was 126 5 in men and 115 in women

It is commonly stated that the blood pressure rises progressively with age That this is not always the case is shown

by Treadgold's (1933) observations His paper includes a detailed analysis of the blood-pressure readings in 2,497 fit pilots of the Royal Air Force He found that, in these men, there was little change in the systolic pressure between the

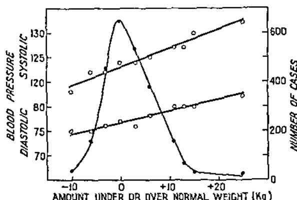


Fig 39 Variation in blood pressure with body build (From figures published by Treadgold)

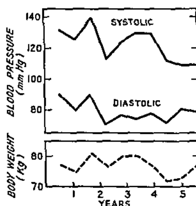


Fig 40 Variation in blood pressure associated with variation in weight in the same individual (Treadgold)

ages of 18 and 40 Treadgold also showed that, in men of normal body-build, the blood-pressure did not vary with height or weight In those who were over-weight or under-weight, on the other hand, both the systolic and diastolic pressures varied with the weight (Fig 39) The same applies to the individual (Fig 40), when he puts on weight and so alters

his body-build—his blood-pressure tends to rise, and vice versa

For insurance purposes it is important to know the average normal blood-pressure at different ages, for the expectation of life is reduced in subjects with a persistently raised pressure Hilton (1936) states that a rise of 30 mm of mercury above the average systolic value about doubles the ratio of actual to expected deaths, and a rise of 50 mm above the average increases this ratio to four times He found that a third of these premature deaths were due to cardiac failure

The average normal values for the systolic pressure given in the actuarial statistics of two insurance companies whose figures I have consulted are in fairly close agreement According to one company they rise from 123 mm Hg at 20 years of age to 131 Hg at 50 The corresponding figures given by the other company are 125 and 135 These insurance statistics indicate that, in the general population, the average systolic pressure does rise year by year instead of remaining practically constant, as Treadgold (1933) found between the ages of 21 and 40 This discrepancy may be due to the fact that Treadgold was dealing with exceptionally fit men, whereas the more sedentary individual tends to lose his physical fitness as he gets older, in accordance with the well-known saying that 'a man is as old as his arteries' Treadgold, however, states that even among fit pilots of the Royal Air Force there are 6 per cent with a systolic pressure of 140 and over

At the other extreme Treadgold found that hypotension might be compatible with perfect health, 3 per cent of fit pilots being hypotensive

Ellis and I (1931) reached the same conclusion from studying athletes at the Olympic Games in Amsterdam in 1928 Four of the twenty-eight Marathon runners examined, during the course of their training, had a diastolic pressure below 70,

and one man, whose systolic pressure was only 105 and diastolic pressure 70, came in third in the race

The problem of hypotension is considered in greater detail by Rook and Dawson (1938), who regard systolic pressures between 110 and 100 or diastolic pressures between 70 and 60 as indicating moderate hypotension, and systolic pressures below 100 or diastolic below 60 as severe hypotension They point out that hypotension may be either occasional or persistent, and that, although persistent hypotension is relatively rare and is usually secondary to some general disease, it may be compatible with perfect health

In a later chapter (p 115) I shall have occasion to refer to the important question of high blood-pressure

I am often asked 'What is the clinical significance of the systolic and diastolic readings?' My answer is that the systolic is a measure of the mechanical efficiency of the left ventricle, whereas the diastolic indicates the resistance against which it has to work Expressed in terms of commerce, these two readings represent the assets and the liabilities of the heart Just as a falling income and a rising expenditure is bound in time to lead to bankruptcy, so a falling systolic pressure and a rising diastolic indicates that failure of the circulation cannot be long postponed

ARTERIAL EXTENSIBILITY

If you examine under the microscope sections of healthy arteries from different parts of the body, you cannot help being struck by the extent to which they differ in structure, in some muscular tissue predominates, in others elastic tissue The reason for these differences becomes evident when one considers the functions of the arteries The muscular arteries, as exemplified by the radial or lingual, serve as taps which regulate the blood-supply to the different organs of the body in accordance with their varying requirements during rest

and activity, whereas the aorta and its main branches form a great elastic reservoir, in which a small increase of pressure suffices to produce a large increase in volume, thereby lightening the work of the heart in discharging its contents, and converting the intermittent cardiac output into a continuous capillary flow

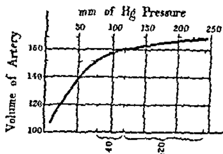


Fig 41 Curve exhibiting relation of total volume of an artery (in arbitrary units) to pressure of contained blood

The extensibility of arteries cannot be measured directly in the living subject, but it can be calculated from observations on the velocity of transmission of the pulse-wave (Bramwell and Hill, 1922) From similar observations on isolated human arteries, removed *post mortem*, it is possible to calculate the extensibility of the arterial wall at different pressures and to plot a curve (Fig 41) showing the increase in volume of an artery for any given increase in pressure (Bramwell, Downing, and Hill, 1923) At low pressures the volume of the artery increases rapidly as the pressure rises, but at high pressure it increases much more slowly, for, at high pressures, the inextensible components of the arterial wall come into play This fact is of considerable clinical importance, for, as will be seen from Fig 41, when the diastolic pressure is 80 a systolic pressure of 120 (i e a pulse-pressure of 40) produces an increase in volume of the artery of about 8 per cent When, however, the diastolic pressure is 120, the arterial wall is already under considerable tension at the end

of diastole, and is much less extensible, hence a pulse-pressure of about 120 (i e three times as great) is required to produce a similar increase in volume When the diastolic pressure is very high, even an enormous pulse-pressure will enable the aorta to accept only a relatively small ventricular output Then, apart from the danger of rupturing diseased

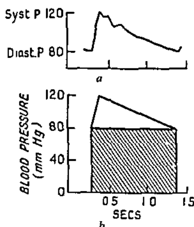


FIG 42 (*a*) Normal sphygmogram (*b*) Absolute sphygmogram from same case

arteries in the brain by excessive pressure or of imposing an intolerable burden on the heart, the limitation of the cardiac output so reduces the oxygen supply to the muscles that sustained physical effort becomes impossible

I referred above (p 59) to the sphygmogram as a 'Pressure-Time Curve' That is not strictly correct It is an 'Expansion-Time Curve', for, owing to the diminution in extensibility of the artery as the pressure rises, a given excursion near the top of the upstroke of the sphygmogram represents a much greater increase in pressure than an equal excursion near the bottom

A more accurate picture of the pressure variations of the pulse is given by what has been termed the 'Absolute Sphygmogram' This is a diagram (Fig 42) in which the

ordinates represent pressure as measured by the *sphygmomanometer* and the abscissae time as shown in the sphygmogram It consists of a rectangle representing the *constant* (diastolic) element of the pressure in the artery, and a superimposed triangle representing the changing (pulse) *pressure* The latter corresponds to the clinical sphygmogram, but depicts pressure instead of volume changes

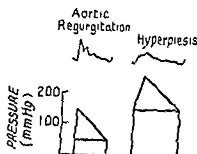


FIG 43 Abnormal sphygmograms with corresponding 'absolute sphygmograms

In Fig 43 two clinical sphygmograms with the corresponding absolute sphygmograms are given for the sake of comparison That on the left is typical of aortic regurgitation, while that on the right is typical of hypertension These two diagrams serve to illustrate the difference in behaviour of the arterial wall when the initial (diastolic) tension is high and low respectively

Arteriosclerosis 'Arteriosclerosis' merely means stiffening of the arteries Even in perfectly healthy people the arteries tend to lose their elasticity with advancing years, they become less extensible, and consequently an increased pulse-pressure is necessary to enable the heart to maintain its output This is clearly seen in old age Thompson and Todd (1922) studied 102 healthy old men (Chelsea pensioners) aged 75–92 The mode in this series was 150/80 The systolic reading is not very high, but the pulse-pressure is raised

One is accustomed to think of arteriosclerosis as a disease

of old age, but, in fact, the arteries begin to lose their elasticity early in childhood Hill, McSwiney, and I (1923) made some observations on this subject in a series of 74 normal

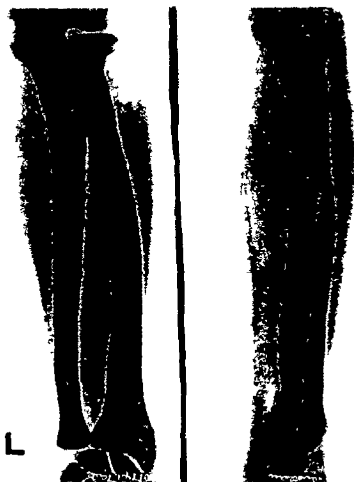


Fig 44 Radiogram of limbs showing calcification of arteries

people We found that the arterial extensibility, as expressed in terms of the percentage increase in volume per mm increase of pressure, diminished progressively from 0 47 at the age of 5 to 0 17 at the age of 80 (Table V) Between the ages of 10 and 60 arterial extensibility is halved

TABLE V

Age	*5*	*10*	*15*	*20*	*30*	*40*	*50*	*60*	*70*	*80*
Mean velocity (Metres per sec)	5 2	5 55	5 9	6 2	6 75	7 2	7 6	8 0	8 3	8 55
Mean extensibility	0 47	0 41	0 36	0 33	0 28	0 24	0 22	0 20	0 18	0 17

Arteriosclerosis is the clinical manifestation of three distinct pathological entities Of these the most important is atherosclerosis, which starts as a fatty degeneration of the intima and later spreads to the media It occurs in patches which may calcify and peel off, leaving ulcers, and affects especially the aorta, the coronary and the cerebral arteries, but not the muscular arteries of the limbs

The second cause of arteriosclerosis is hypertrophy of the muscular elements of the arterial wall This is a particularly interesting lesion, since it represents the physiological response to high blood-pressure, and is not only analogous, but proportional, to the hypertrophy of the left ventricle found in this condition In fact Turnbull (1914) states that, *from microscopical examination of the hypertrophied arteries,* it is possible to estimate the increase in the weight of the heart

Lastly, calcification of the media may convert the artery into a rigid tube which is opaque to X-rays (Fig 44) Unlike atherosclerosis, hypertrophy and calcification of the muscular coat tend to occur in the arteries of the limbs

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CHAPTER V

CARDIAC ARRHYTHMIAS

REPEATEDLY in the history of science the invention of a new instrument, or a new method of investigation, has determined the lines along which knowledge shall advance The introduction of the stethoscope by Laennec, in 1819, interested physicians in heart-murmurs and focused attention on lesions of the heart valves Thirty years later the invention of the *sphygmograph* provided a graphic method of recording the pulse and opened up new possibilities of studying irregular heart action

This instrument enabled us to distinguish between various types of irregularity of the pulse, particularly that in which the beats were completely irregular both in time and force, and that in which the heart's action was merely intermittent, that is to say, the regular rhythm was from time to time interrupted by a pause, two pulse-beats being separated by double the normal interval What it failed to tell us was the nature of the disturbance responsible for the arrhythmia

In 1902 Mackenzie introduced the *clinical polygraph*, which gave synchronous records of the venous and arterial pulses This made it possible to study the time relations of auricular and ventricular systole

Mackenzie was a very remarkable man most of his working life was spent in general practice in Burnley, nevertheless, during those busy years his boundless energy enabled him to find time for original work and to make contributions to knowledge, both clinical and physiological, which earned him world-wide reputation as a master of his craft While practising in Burnley, he spent many happy hours in the Manchester Royal Infirmary, going round the wards and

discussing clinical problems with his friend Graham Steell, for whom he had a high regard

The venous pulse in the neck can be distinguished from the arterial pulse by its position, by its character, and by the fact that, being due to a change in volume with very little change in pressure, it is visible but not palpable

When, however, the tricuspid valve is incompetent the right ventricle drives blood back into the veins of the neck and

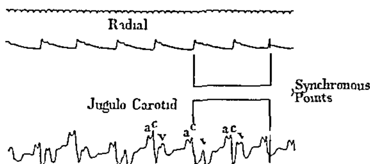


FIG 45 Polygraphic record of normal venous pulse

into the liver, and the venous pulse becomes easily palpable in these two situations Similarly, when the tricuspid orifice is stenosed, the right auricle hypertrophies, and when it contracts it drives some of the blood back under considerable pressure into the neck veins

The normal venous pulse (Fig 45) exhibits three waves known as '*a*', '*c*', and '*v*' The '*a*' wave is due to the rise of pressure in the jugular vein produced by auricular systole, and the '*c*' wave is synchronous with and partly attributable to transmitted pulsation from the adjacent carotid artery Thus the time interval between '*a*' and '*c*' is a measure of the interval which elapses between the commencement of auricular and ventricular systole The '*v*' wave is due to back pressure as the blood accumulates in the auricle behind the closed tricuspid valve

Mackenzie found that in the condition we now call

auricular fibrillation, in which the pulse is completely irregular, no '*a*' wave was present on the venous tracing, and this led him to conclude that the auricle had failed to contract He also showed that there were two types of intermittent pulse In the one, which is due to heart-block, there was no '*c*' wave on the venous curve, the ventricle having failed to contract, in the other the '*c*' wave was premature In the latter the ventricle contracts early in diastole, before it has had time to fill with blood, and consequently its output is so small that no pulse-wave or only a diminutive pulse-wave reaches the wrist Another type of irregularity differentiated by the polygraph was 'sinus arrhythmia', in which the three waves of the venous tracing exhibit the normal sequence, but there is a phasic quickening and slowing of the pulse This often coincides with the phases of respiration, and can then be accentuated by deep breathing

The clinical polygraph has now been superseded by the *electrocardiograph*, the invention of a Dutch physiologist—Einthoven Shortly after the First World War I attended a meeting of the Physiological Society in Einthoven's laboratory in Leyden—the first meeting of the Physiological Society, in the fifty years of its existence, held outside Britain That Leyden should have been chosen for its meeting-place is some indication of the respect in which Einthoven was held by British physiologists

The electrocardiogram enables us to follow the progress of the wave of excitation in the heart from its starting-point in the sino-auricular node, through the muscular tissue of the auricle to the auriculo-ventricular node, and thence through the A-V bundle of His and its branches which ultimately supply twigs to every muscle fibre in the ventricles (Fig 46)

In its early days electrocardiography was used chiefly to distinguish between the different types of cardiac arrhythmia and, for this, the three standard leads (lead I, R hand to

L hand, lead II, R hand to L leg, lead III, L hand to L leg) were adequate That chapter of the story is virtually closed, for, thanks to what electrocardiography has taught us, we can now recognize all the common types of cardiac arrhythmia by the unaided senses

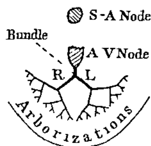


FIG 46 Diagram of neuromuscular conducting mechanism S–A Node = sino-auricular node, A–V Node = auriculo ventricular node R and L = right and left branches of the auriculo ventricular bundle

Today, electrocardiography is concerned chiefly with the investigation of myocardial infarction For this purpose it was found that leads, in which one electrode is attached to the R arm or L leg and the other (the 'exploring electrode') is placed directly on the surface of the chest, may yield information not given by the standard leads When the exploring electrode is placed over the region of the cardiac apex this is called lead IV More recently Frank Wilson (1944) has shown that much more accurate information concerning lesions of the myocardium can be obtained from 'unipolar' leads, in which the indifferent electrode is connected through a resistance to all three instead of to only one limb, while the exploring electrode is applied to a series of points on the chest wall, or attached to a limb Wilson's unipolar leads have revolutionized electrocardiography, but, for the diagnosis of the cardiac arrhythmias, they are unnecessary and so I propose to confine my attention to the three standard leads

The normal electrocardiogram consists of a series of deflexions named after the letters of the alphabet, *P*, *Q*, *R* *S*, *T*, and *U* *P* is associated with excitation in the auricles, *Q*, *R*, and *S* with the rapid spread of excitation to all the muscle fibres of the ventricles, and *T* with the gradual subsidence of the state of excitation in the ventricles The significance of *U* is still undetermined Thus, the first rapid ventricular

deflexion marks the beginning, and *T* the end of ventricular systole

There is a wide range of variation between electrocardiograms from different normal subjects (Fig 47), but all have

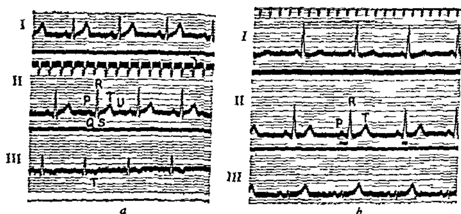


FIG 47 Normal electrocardiogram Time marker = 0 2 sec
(a)
(b)

certain features in common, namely, the time-relations of the different deflexions and their form In health the time interval between the commencement of auricular systole and ventricular systole, as represented by the interval between the commencement of *P* and the commencement of the first rapid ventricular deflexion, is never greater than 0 2 sec Similarly the time taken by the excitation wave to activate all the muscle fibres of the ventricles, as represented by the initial group of rapid ventricular deflexions, 'the *QRS* complex , is never greater than 0 1 sec In normal records, in the standard leads I and II, *P* and *T* are always positive, though in lead III they may be negative

AURICULAR FIBRILLATION

Faradic stimulation of the exposed auricle of a dog brings the normal cardiac mechanism to a standstill co ordinated

auricular contraction ceases and is replaced by faint quivering movements involving small isolated groups of muscle fibres in the quiescent auricular wall This we call auricular fibrillation At the same time the ventricular rate becomes much more rapid and its rhythm completely irregular

In the normal heart the 'pace-maker' (the sino-auricular node) generates rhythmic stimuli in an orderly manner, at a rate of about 70 per minute, and each stimulus causes contraction first of the auricles and later of the ventricles In auricular fibrillation, the normal pace-maker is out of action, there is no co-ordinated contraction of the auricles, and stimuli are showered upon the ventricles at a rate which may be as high as 500, or more, per minute It is impossible for the ventricle to contract in response to all these stimuli, but its rate may increase up to about 180 per minute, while its rhythm becomes completely irregular Consequently, from time to time, two beats follow each other in rapid succession When this happens, the ventricle contracts before it has had time to fill with blood Such beats are unproductive on account of the small output of the heart, and only a very feeble pulse-wave, or it may be no pulse-wave at all, reaches the periphery Thus the heart-rate, as counted with the stethoscope at the apex, is faster than the pulse-rate at the wrist the difference between the two counts is called the 'pulse deficit' The larger the number of unproductive beats, the greater is the pulse deficit, and the more useless work is the ventricle required to perform It is merely tiring itself out to no purpose

Students are apt to get the impression that the feeble pulse-beats, felt at the wrist, are due to incomplete contraction of the ventricle That is not so, they are due to incomplete filling of the ventricle during diastole

One of the hypotheses suggested to account for the mechanism of fibrillation is based on the experimental observations of

Mines (1913) who showed that, when a ring of muscular tissue is stimulated by a single induction shock, one of two things may happen Generally, a wave of contraction starts on either side of the point stimulated (Fig 48*a*) The two waves travel in opposite directions and, when they meet on the far side of the ring (Fig 48*b*), each is brought to a standstill, since the muscle in front of each advancing wave has recently contracted and is, for the time being, incapable of responding

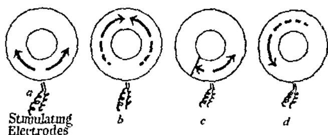


FIG 48 Diagram to illustrate circus movement

to a second stimulus Occasionally, however, one wave fails to start (Fig 48*c*) The other then completes the circuit of the entire ring (Fig 48*d*) and on reaching its starting-point, finds the muscle fully recovered from the previous contraction and so is able to continue to travel round and round the ring indefinitely To this phenomenon Mines gave the name 'circus movement'

Garrey (1914) working independently in America observed the same phenomenon and suggested that this might be the mechanism underlying auricular fibrillation, a suggestion which some eight years later was confirmed by the work of Lewis and his associates at University College

If, at any time, the circulating wave finds the muscle in front of it still refractory (*i e incapable of responding to* stimulation), it will be brought to a standstill, and the normal pace-maker will have *a chance to reassert itself* This would happen if the refractory period were prolonged, and is what

we are attempting to achieve when we prescribe quinidine Unfortunately quinidine also slows the rate at which the wave is travelling, so that, in spite of the prolonged refractory period, the muscle may still have time to recover before the wave completes the circuit Whether treatment is or is not successful depends on which of these two actions predominates

Digitalis, the drug which we generally use in the treatment of auricular fibrillation, has quite a different action from quinidine The beneficial effects produced by digitalis in the treatment of dropsy were first described by the great Birmingham physician William Withering, in 1785 So accurate were his observations, that it would be hard to improve upon the masterly description that he gave more than 150 years ago. Digitalis does not stop fibrillation, it merely prevents some of the stimuli from the auricle reaching the ventricle and so slows the ventricular rate The two sphygmograms in Fig 49 were taken, the one before, and the other after, treatment with digitalis In the first record many of the beats are of very small amplitude, in the second record all but one of the small, unproductive, beats have been eliminated

These two drugs differ in another way Quinidine is rapidly eliminated from the body, so in order to produce the full therapeutic effect we must give repeated doses at short intervals of time in practice we give it two-hourly Digitalis, on the other hand is a cumulative drug, that is to say its rate of clearance from the body is extremely slow, so the usual procedure is to start with one grain of the powdered leaf two or three times a day With this dose the full therapeutic effect will not be obtained for several days, but there will be no danger of producing toxic symptoms In exceptional cases when the patient s condition is so grave that rapidity of action is essential much larger doses may be given, but under such circumstances in order to avoid overdosage from

cumulative action, one must be sure that the patient has not taken any digitalis for the previous two or three weeks In cases of urgency, we give the drug intravenously, in the form of digoxin (1 0 mgm)

It is important to remember that there is a wide range of variation between different individuals in their tolerance for

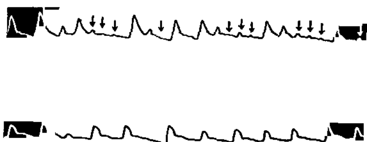


FIG 49 Sphygmograms from a case of auricular fibrillation Upper record before treatment showing complete irregularity and a large number of very small pulse waves (marked with arrows) Lower record after treatment with digitalis showing greater uniformity in rhythm and form of pulse most of the small ineffective beats having been eliminated

certain drugs Clark (1939) states that, in the case of digitalis, this probably amounts to at least 400 per cent Thus a dose which is insufficient to produce benefit in one patient may prove toxic to another who is unusually susceptible For that reason, when using powerful drugs, it is always advisable to proceed with caution, starting with a moderate dose and increasing it gradually

With all drugs there are certain well defined boundaries to the region in which we work—the 'therapeutic range' If we transgress the upper limit, toxic symptoms develop while, if we are too faint-hearted we may fail to reach the lower limit In my experience, when prescribing digitalis, the latter is the more common mistake

When the patient is fully digitalized, only a maintenance dose (i e a dose sufficient to balance the amount eliminated or destroyed in the body) will be required An excessive

maintenance dose given over a long period is the most common cause of digitalis poisoning

Closely related to auricular fibrillation is the condition known as Auricular Flutter This disorder also is believed

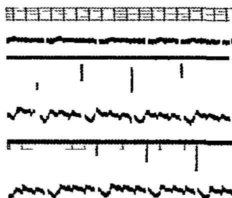


Fig 50 Electrocardiogram showing auricular flutter in which the block is for the most part 4 1

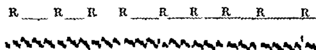


Fig 51 Auricular flutter with a varying ventricular response

to be due to circus movement in the auricle but the excitation wave circulates at a much slower rate than in fibrillation generally in the neighbourhood of 250–300 per minute and co ordinated contraction of the auricle occurs In most untreated cases of flutter the ventricle responds to every second auricular impulse so that the heart rate is in the neighbourhood of 125–150 The degree of block at the junctional tissues may be increased by digitalis to 4 1 instead of 2 1 (Fig 50) giving a pulse rate of about 60–75 per minute with a corresponding increase in cardiac efficiency Sometimes the pulse in flutter is irregular owing to a varying degree of A V block (Fig 51)

SINUS ARRHYTHMIA

This (Fig 52) is the commonest type of irregular heart action It is not pathological In the experimental animal vagal stimulation slows the rate of impulse production in the S–A node The vagus is concerned also with the reflex



FIG 52 Electrocardiogram Lead II showing sinus arrhythmia

control of respiration and, in healthy children and young people, it is not uncommon to find that the heart-rate slows during expiration and quickens during inspiration This phasic type of variation may persist in later life and then, in my experience, is not infrequently associated with a tendency to fainting

EXTRA-SYSTOLE

Stimulation of the exposed ventricle by a single induction shock gives rise to what physiologists call a 'forced beat' This beat is independent of the rhythmic sequence of contractions in response to stimuli coming from the S–A node The same thing occurs spontaneously in man These premature beats are commonly known as 'extra-systoles' It is a misleading term, because it is only occasionally that they are actually supernumerary In most cases the premature beat is followed by a prolonged diastole, so that the combined duration of the cycles preceding and following the premature beat is equal to two normal cycles

In Fig 53 the first three cycles are normal, auricular systole being followed by ventricular systole, but the fourth ventricular complex is premature, it occurs in response to a stimulus initiated by the ventricle, not by the S–A node, and is

comparable to a forced beat produced by stimulating the ventricle The ventricular complex of this premature beat differs from that of the normal beats in the prolonged duration of the initial ventricular deflexion (*R*) This is due to the fact that the ventricle which initiates the premature beat contracts

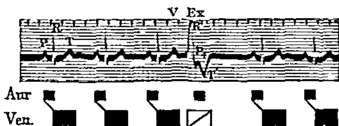


FIG 53 Electrocardiogram showing ventricular extra systole (V Ex) The diagram illustrates the sequence of auricular (Aur) and ventricular (Ven) contractions The fourth auricular complex (P_4) appears as a notch in the *T* wave of the extra systole
■ = normal beat ◻ extra systole

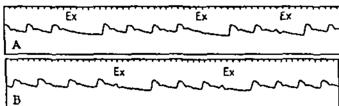


FIG 54 Radial sphygmograms showing normal rhythm interrupted by extra systoles (Ex)

immediately, but, in order to reach the other ventricle, the stimulus has to spread either through the septum or up one branch of the bundle and down the other, thus excitation is delayed The rhythmic auricular impulse (P_4, Fig 53) which follows the premature beat reaches the ventricle while it is still refractory and it is not until the succeeding rhythmic impulse arrives that the ventricle again contracts, hence the 'compensatory pause'

The more premature the extra systole, the shorter is the preceding diastole Consequently ventricular filling is defective and only a very feeble pulse wave, or it may be no pulse-wave at all, reaches the periphery (Fig 54) Conversely,

during the prolonged diastole following an extra-systole, the ventricle is over-filled and the pulse-beat following the pause

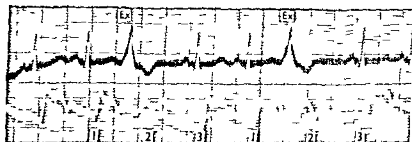


FIG 55 Synchronous electrocardiogram and carotid sphygmogram to show
1 Normal beat.
2 Small volume of beat due to extra systole (Ex)
3 Large volume of beat following extra-systole

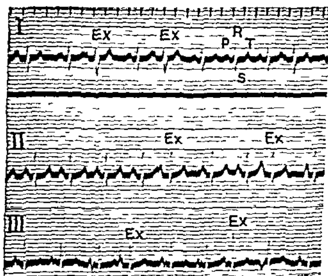


FIG 56 Electrocardiogram showing supraventricular extra-systoles (Ex) in which the ventricular complex is slightly aberrant

is of increased amplitude (Fig 55) The patient may be conscious of this thumping beat or of the pause preceding it One of my patients, in whom every fourth beat was premature, compared the sensation to being in a rowing-boat in a rough sea. He felt as if he were being suddenly lifted up

on the crest of a wave and then, just as the boat righted itself, it struck another wave and shot up again Clinically, an intermission in the pulse due to an extra-systole can be recognized on auscultation, for, during the intermission, the

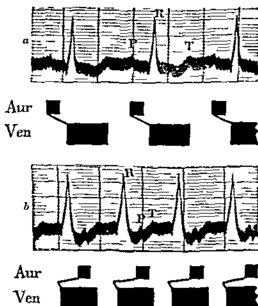


FIG 57 Two electrocardiograms from the same case The upper record shows normal rhythm the lower record part of a paroxysm of nodal tachycardia Aur auricular systole Ven — ventricular systole

sounds of the premature beat are heard, following closely upon those of the preceding normal beat

When the premature beat is initiated not by the ventricle but by the auricle, the impulse reaches the ventricle through the normal channels, and consequently the ventricular complex in the electrocardiogram is of the normal form (Fig 56)

Not infrequently the stimulus which initiates an extra-systole arises in the A–V node It then spreads upwards to the auricle and downwards to the ventricle (Fig 57), reaching the two chambers almost simultaneously, but when the ventricle contracts, the tricuspid valve closes and the auricle,

being unable to empty itself into the ventricle, drives some of the blood back into the veins of the neck Forcible venous pulsation in the neck, due to this cause, is sometimes a troublesome symptom in a condition to which I shall now refer—'Paroxysmal Tachycardia'

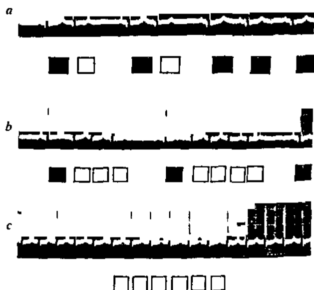


FIG 58 Electrocardiogram from same case as Fig 59 (*a*) single extra systoles (*b*) groups of extra systoles (*c*) part of paroxysm of tachycardia ■ – Normal beat □ – Extra systole

The three records in Fig 58 were taken from a patient with nodal extra-systoles In the first record (*a*), the first and third cycles are each followed by an extra systole but the last three cycles are normal In the second record (*b*), instead of a single premature beat following the normal cycle, there are groups of three or four premature beats occurring in rapid succession The third record (*c*), which was taken a few minutes later, consists of a long series of premature beats without any intervening normal cycles It is what we call a 'paroxysm of tachycardia' These three records illustrate the relationship of extra-systoles to paroxysmal tachycardia

Fig 59 is a sphygmogram showing a short paroxysm of tachycardia preceded by a spell of irregular heart action, due to multiple extra systoles, and followed by a run of normal rhythm

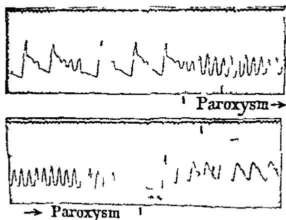


FIG 59 Sphygmogram showing short paroxysm of tachycardia

Paroxysms of tachycardia occur quite independently of exertion or any other obvious provoking cause, and are characterized by the abrupt onset and termination of the tachycardia

HEART-BLOCK

In 1883 Gaskell, a great physiologist who still lectured to us at Cambridge when I was an undergraduate, showed experimentally in the tortoise that, by vagal stimulation, or by compression of the tissues joining the auricle and ventricle, it is possible to delay, or to prevent, the passage of stimuli from the one chamber to the other Subsequently Erlanger (1905), in whose laboratory I had the privilege to work in 1923–4, was able to produce varying grades of heart block in dogs by clamping the A–V bundle

Fig 60 is a sphygmogram from a patient with heart-block The pulse is intermittent, that is to say the dominant rhythm is from time to time interrupted by a pause of twice the

normal length, but unlike the intermission due to an extra-systole, no heart-sounds are audible during the pause, for the ventricle has failed to contract

The mechanism *of heart-block is clearly shown* by the electrocardiogram (Fig 61) In the first cycle the *P–R* interval (as indicated by the line under the record) is less than 0 2 sec ,



Fig 60 Sphygmogram showing intermittent pulse due to partial heart block with dropped beats

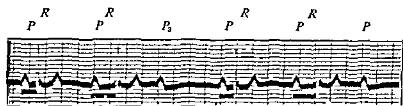


Fig 61 Electrocardiogram Lead II from a case of partial heart block with dropped beats Lines under record indicate duration of *P–R* interval

in the second cycle it is almost 0 3 sec , while the third impulse *leads to contraction of the auricle, as shown by the* deflexion P_3, but fails to reach the ventricle This sequence of events is repeated in the next three cycles A record such as this gives a remarkably vivid picture of what is taking place within the heart The series of impulses passing from auricle to ventricle is like a convoy of wagons crossing a muddy field, each wagon makes the ruts deeper, the going becomes heavier and heavier, until eventually the track is no longer *passable, and the next wagon has to wait for it to be repaired*

In 'Complete Dissociation' (Fig 62), none of the auricular impulses succeed in passing the junctional tissues and the ventricle beats with a rhythm of its own which bears no relation to that of the auricles Such an 'idio-ventricular rhythm' can generally be recognized clinically by its slow rate it is generally under 40 per minute and sometimes under 30

When the block is situated, not between auricle and ventricle, but distal to the A–V node, in one of the branches of the bundle of His, the excitation wave has to travel down the



FIG 62 Electrocardiogram showing complete auriculo ventricular dissociation
In diagram Aur = auricular Ven — ventricular systole

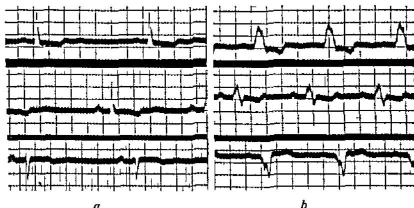


FIG 63 (*a*) Normal record (*b*) Bundle branch block Note spreading and notching of QRS complex The notching is due to asynchronous excitation in the two ventricles

normal branch of the bundle and through the muscular tissue of the septum in order to reach the affected ventricle Thus excitation is delayed, as shown by the increased duration of the *QRS* complex in the electrocardiogram, which resembles that of a ventricular extra-systole The two records in Fig 63 were taken from the same patient the first shows normal complexes and the second 'bundle branch block'

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CHAPTER VI

HEART FAILURE

THE term 'Circulatory Failure' is generally understood to signify a condition in which the output of the heart is not sufficient to meet the requirements of the body, even at rest This may be the result either of damage to the heart which renders it mechanically incapable of maintaining its output, or of deficient venous filling When the venous return to the heart is reduced, owing to stagnation of blood in the capillary area, as in shock, we speak of the condition as 'peripheral failure', the term 'heart failure' being reserved for those cases in which the heart itself is at fault

Heart failure is not synonymous with a reduction in cardiac output, it represents rather a deficit in the balance between supply and demand The requirements of the body vary greatly in different conditions In thyrotoxicosis, for example, the increased metabolism makes a greatly increased demand upon the heart and heart failure may appear even when the cardiac output is well above the average normal figure

Exercise tolerance The healthy heart is able not merely to satisfy the requirements of the body at rest, but greatly to increase its output to meet the additional demand for oxygen of the active muscles during physical exertion Its capacity to do so is called the 'cardiac reserve' The athlete increases his reserve by training, whereas the person who leads a sedentary life has a reduced reserve through being 'out of training' Thus the tolerance for exercise is a measure of the cardiac reserve, and the most reliable guide to a patient's exercise tolerance is an accurate clinical history I always ask recruits for the Services, 'Do you play games?' 'What is the nature of your work?' and 'Do you believe yourself to be perfectly

fit?', for, if a man plays football every week-end or if he can follow an occupation involving severe physical exertion, there cannot be much wrong with his heart Physical efficiency tests (see p 55) are only necessary in exceptional circumstances

Limitation of the tolerance for exercise is not always due to disease of the heart or lungs, it also occurs in neurocirculatory asthenia (see p 4)

We are accustomed to think of people as either fit or unfit, but there are many degrees of fitness, healthy people vary greatly in their capacity for physical exertion, some are robust and athletic, others are only suited to a more sedentary mode of life A random sample of the population yields a series of types intermediate between the athlete and the patient with heart disease, the athlete is at the top of the ladder, the patient with heart failure at the bottom, most of us get about half way up, by training we can mount a few steps higher, but if we neglect our health we drop down lower

In addition to variations due to physique, there are the limitations imposed by age Men in the sixties often wrongly imagine that they are suffering from heart disease, because they find that they are breathless when they attempt to do things which previously they could accomplish with ease They fail to realize that their symptoms are due, not to disease, but merely to advancing years Thus rightly to assess impairment of the cardiac reserve produced by disease, numerous factors such as age, habits, and mode of life must all be taken into account, for each of these takes its toll before disease steps in One must compare the individual patient, not with an arbitrary fixed standard, but with the standard which he himself has set up, as indicated by the mode of life he had been accustomed to lead, prior to his illness Was it active or sedentary? Is he no longer able to do what he did previously?

Cerebral anaemia resulting from a reduced cardiac output may cause fatigue and lassitude, but the most significant manifestations of impending heart failure are dyspnoea and pain

Dyspnoea In Fig 64, I have represented the relation of pulmonary ventilation to physical work *N* is the curve of the

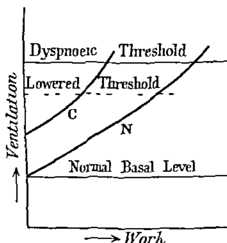


FIG 64 Diagram to illustrate relation of pulmonary ventilation to physical work *N* = normal subject *C* cardiac patient

normal subject it does not start from the base line, because, even at rest, oxygen is required to meet the basal requirements of the body Physical work makes a further demand on the pulmonary ventilation, which increases, at first slowly, and then more rapidly, until eventually the subject becomes conscious that he has to make an effort to get his breath This we call 'dyspnoea' or laboured breathing In the corresponding curve *C* from the patient with heart disease, since the basal requirements are greater than in health, the curve starts at a higher level, it rises more steeply and reaches the threshold of dyspnoea earlier than that of the healthy individual But there is another factor which must be taken into account in assessing subjective symptoms such as dyspnoea, namely, the

nervous threshold Some people become conscious of discomfort earlier than others In highly strung, as opposed to phlegmatic individuals, *the threshold of dyspnoea is lowered*, whether the heart be diseased or healthy

Pulmonary ventilation is controlled by the respiratory centre which is affected by both nervous and chemical stimuli In patients with heart disease the former, but in healthy individuals the latter, play the more important part The nervous stimuli may be vagal reflexes from the congested lungs, or may come directly from the higher centres of the brain the chemical control of respiration depends on the variation in the hydrogen-ion concentration of the blood

The actual process of muscular contraction is anaerobic, and depends on the conversion of glycogen into lactic acid with the liberation of energy This is followed by what is called the 'recovery phase', during which lactic acid is disposed of, partly by oxidation to carbon dioxide and partly by reconversion into glycogen During exercise, the contraction and recovery processes take place concurrently, and so long as the removal of lactic acid keeps pace with its production, an equilibrium is established and exercise can proceed As soon, however, as the oxygen-supply becomes inadequate to provide for oxidation of the lactic acid, this substance accumulates in the tissues and, as A V Hill and Lupton (1922–3) expressed it, the body 'goes into debt' for oxygen The panting which follows the cessation of exercise is necessary to pay off this debt, by supplying sufficient oxygen to provide for the removal of the lactic acid which has accumulated in the body during exercise In patients with heart disease, however, *the most important factor in the production of dyspnoea is pulmonary congestion* This reduces the vital capacity of the lungs and the tolerance for exercise

Paroxysmal dyspnoea Sometimes dyspnoea occurs spontaneously, apart from exertion, and may even wake the

patient from sleep This is known as 'Paroxysmal Dyspnoea' or 'Cardiac Asthma', and is due to sudden congestion in the pulmonary circuit, which is recognized clinically by the presence of moist sounds at the pulmonary bases and radiologically by increased density of the vascular shadows in the lungs This type of dyspnoea is liable to occur in patients in whom failure of the left ventricle is associated with a normally acting right ventricle, while the latter maintains the input of blood to the lungs, the former is unable to keep pace and pass the blood on to the systemic circuit In most cases of heart failure both right and left ventricles are involved, but, when there is systemic hypertension or a lesion of the aortic valve, the strain falls primarily on the left ventricle

Dyspnoea unrelated to exertion may occur in patients with uraemia or diabetes owing to a disturbance of the acid-base balance

Patients often say that they are 'short of breath', when what they really mean is that they find it difficult to take a full breath, or as they sometimes express it 'to get to the bottom of their breath' In such cases the patient may be observed to sigh for no apparent reason This irregular sighing type of respiration is a manifestation not of heart disease but of general nervous exhaustion It is much more common in women than in men and is especially prone to occur in those of a highly strung nervous temperament The incidence of this symptom has, in my experience, greatly increased in recent years, presumably owing to the prolonged strain of the conditions under which we have been living during and since the war

Vital capacity During quiet respiration about 0 5 l of air passes to and fro through the larynx at each breath This is called the 'tidal air' At the end of expiration the chest still contains about 3 0 l of air Half this—the 'reserve' or 'supplemental air'—can be expelled by a forced deep

expiration, but the other half—the 'residual air'—remains in the lungs Conversely a forced deep inspiration will increase the amount of air taken into the lungs by about 1 5 l This is called 'complemental air' The sum of the reserve, complemental, and tidal air is called the 'Vital Capacity' of the

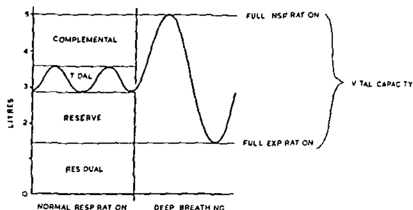


FIG 65 Diagram to illustrate components of vital capacity

lungs and is the volume of air expelled by the deepest possible expiration following the deepest possible inspiration (Fig 65)

Clinically, estimation of the vital capacity is of value not only in respiratory but also in cardiovascular disease, for pulmonary congestion, due either to back pressure in mitral stenosis or to failure of the left ventricle to empty the lungs, reduces both the alveolar volume and the pulmonary distensibility Thus variations observed in serial estimations of vital capacity indicate an increase or a decrease in pulmonary congestion

As in all such tests, it is essential to obtain the intelligent co-operation of the patient, for, unless he fills and empties his lungs as completely as possible, a fallacious reading will be obtained

CONGESTIVE HEART FAILURE

When the output of the right ventricle fails to keep pace with the venous inflow, the venous pressure rises, and we

say that the patient is suffering from 'Congestive Heart Failure'

For research purposes the venous pressure is measured by introducing into a vein a cannula connected to a manometer, or by catheterization of the right auricle Clinically, we recognize raised venous pressure by engorgement of the veins in the neck The normal venous pressure is so low that the veins in the neck collapse if the head and shoulders be raised slightly above the level of the right auricle, but, in congestive heart failure, they remain engorged even when the patient sits up As the pressure in the right auricle rises, the cardiac output increases in accordance with Starling's law, but there is a limit to the pressure the auricle can cope with and, once the maximal output has been attained, a further rise in venous pressure leads to a diminished cardiac output It is at this stage that venesection is beneficial and conversely, transfusion dangerous, since it adds still further to the venous pressure

Other clinical manifestations of congestive heart failure are enlargement of the liver, oedema, and ascites The liver is very sensitive to a rise in venous pressure and enlargement is easily detected by palpation

Pure right-ventricular failure is generally secondary to chronic pulmonary disease or to obstructive lesions in the smaller pulmonary vessels and, in the absence of associated left-sided failure, does not give rise to dyspnoea, since there is no pulmonary congestion In patients with mitral stenosis, on the other hand, breathlessness is the presenting symptom This is due not to left heart failure but to back pressure on the lungs, producing pulmonary congestion

Oedema In congestive heart failure, slowing of the circulation rate results in capillary stasis and malnutrition of the capillary walls, while back pressure from venous engorgement causes a rise in capillary pressure This leads to exudation of fluid from the capillaries into the subcutaneous tissues

There is, however, another very important factor in the production of cardiac oedema, namely, salt retention, due to renal impairment, resulting probably from venous engorgement, Reaser and Burch (1946) have shown that a normal subject can excrete very much more radio-active sodium than a patient with congestive heart failure Salt restriction is accordingly a primary consideration in the treatment of these cases

Oedema occurs in association with many conditions other than heart disease, of which the most common is nephritis Cardiac and renal oedema differ in their distribution In cardiac cases the distribution of the oedema is determined by gravity and consequently it appears first in the dependent parts of the body (i e in the feet and ankles in patients who are up and about, but in the lumbo-sacral region and in the back of the thighs in patients who are bedridden) It is more pronounced in the evening and, at first, is noticed only when the patient has been on his feet all day, disappearing by morning, after the night's rest By contrast, renal oedema may be more conspicuous on rising in the morning and tends to be more generalized in its distribution

The body is capable of retaining a considerable volume of fluid in the tissues before oedema becomes apparent Conversely, the disappearance of oedema does not signify that all the surplus fluid has been eliminated This is clearly shown by a record of the patient's weight

Cyanosis When the circulation rate is reduced, the blood travels more slowly through the capillaries and the utilization of oxygen by the tissues is correspondingly increased Hence there is a higher percentage of reduced haemoglobin in the capillary blood and this first manifests itself by blueness of the lips

In vitro the normal blood, completely saturated, contains 20 volumes per cent of oxygen (i e a litre of blood will take

up 200 ml of oxygen), but, in passing through the lungs, it only gets saturated to the extent of about 19 volumes per cent Normal arterial blood is therefore 1 volume per cent unsaturated A further 5 volumes per cent of oxygen is removed in the capillaries Hence the venous blood is 6 volumes per cent unsaturated We may take the mean desaturation value of the capillary blood as equal to 3 5 volumes per cent (i e midway between the value of arterial and venous desaturation) Lundsgaard (1919) has shown that cyanosis appears when the capillary blood contains 5 gm of reduced haemoglobin per 100 ml , that is to say when it is 6 7 volumes per cent unsaturated, since each gramme of haemoglobin combines with 1 34 ml of oxygen

Apart from increased utilization of oxygen in the tissues, cyanosis may result from defective oxygenation of the blood in the lungs, as in emphysema, or from shunting of the blood from the right to the left heart either directly, as in certain congenital heart lesions (see p 110), or indirectly by passing through unaerated lung tissue, as in pneumonia

Obviously it is only when cyanosis is due to defective oxygenation of the blood in the lungs, that we can hope to achieve much by oxygen therapy, since, in cyanosis due to other causes, the arterial blood leaving the lungs is already fully saturated with oxygen

MYOCARDIAL ISCHAEMIA

When the output of the heart is insufficient to meet the oxygen requirements of the body as a whole, lactic acid accumulates in the tissues and the patient becomes short of breath, but when the defective blood-supply affects an isolated group of muscles the presenting symptom is not breathlessness but pain

MacWilliam and Webster (1923) showed that if a tourniquet be applied to one arm and the subject be told to clench

both fists repeatedly, pain soon develops in the muscles of the forearm on the affected side and makes further movement of the fingers of that hand impossible

Similarly, when the lumen of the arteries of the lower limbs is narrowed by disease and they become like corroded pipes, the blood-supply to the muscles is curtailed, and the patient develops what is known as 'intermittent claudication', after walking a short distance he is pulled up by cramp in the calf muscles, if he walks on the pain becomes more severe, but if he stops and rests it soon passes off and he is able to walk another short distance before the pain recurs Likewise, when the blood-supply to the heart-muscle is reduced by coronary obstruction, the patient's activities are limited by pain in the chest—'angina pectoris'

'Angina of effort' is strictly analogous to intermittent claudication for, after walking a certain distance, the patient complains of pain in the chest, which increases in severity if he persists, but passes off if he rests Less commonly anginal pain occurs at rest as the result of emotional stimuli and, since this pain is generally relieved by vasodilator drugs we believe that it is due to muscular spasm of the coronary arteries If, however, one of the coronary branches be completely occluded by a blood clot, the pain is not relieved by resting, since the area of the heart wall supplied by the affected branch is permanently cut off from its blood-supply and infarction results

Many patients who have suffered from angina of effort ultimately die of coronary occlusion The two conditions are clearly related and have a common pathology, namely coronary atherosclerosis Gilchrist and Hill (1938) submitted a series of patients suffering from angina of effort to an exercise test of sufficient severity to induce a mild attack of pain They took electrocardiograms before and after exercise and were able to show that, in most cases, during the process of

recovery, the electrocardiogram exhibited a series of *T*-wave changes similar to those seen following coronary occlusion, but, whereas in the latter these took days, weeks or even months to develop, in their patients they occurred in a matter of minutes Similar observations have recently been recorded by Paul Wood et al (1950) Morgan Jones and Wade (1949) have produced electrocardiographic evidence that many patients whose symptoms would suggest a diagnosis of angina of effort, actually have a pre-existing cardiac infarct

Myocardial ischaemia, however, may be produced by various conditions other than coronary atherosclerosis—it may be due to obstruction of the mouths of the coronary arteries by syphilitic aortitis, or merely to low pressure in the coronary circuit The heart differs from all other organs of the body in that its nutrition depends not on the systolic but on the diastolic pressure During systole the blood is squeezed out of the coronary vessels, and it is only during diastole that it can pass freely through them, hence it is the diastolic pressure which determines the effective filling of the coronary system In patients with free aortic regurgitation the diastolic pressure may be very low, hence these patients are liable to suffer from myocardial ischaemia and cardiac pain Likewise, during a paroxysm of tachycardia, owing to the diminished cardiac output, the coronary pressure may fall sufficiently to produce myocardial ischaemia Lastly ischaemia of the myocardium may be due to deficiency of haemoglobin in the blood itself and patients with severe anaemia are liable to suffer from angina, though there is reason to believe that, in such cases, the anaemia is merely an aggravating factor and that the patient will not suffer from cardiac pain unless there be associated coronary disease

The discovery that cardiac pain is due to myocardial ischaemia has led to an unfortunate confusion of terminology Angina pectoris has long been recognized as a very

important form of heart disease and was, at one time, regarded as a distinct clinical entity; but, if we apply the term '*angina*' to pain produced by any of the various pathological processes which give rise to myocardial ischaemia, it can have no place in the aetiological classification of disease, it is merely a symptom For that reason I prefer to reserve the time-honoured title of 'angina' for the disease due to coronary atherosclerosis, which may manifest itself either by angina of effort or by coronary occlusion, and not to apply it to the other conditions in which cardiac pain is merely a symptom

MacWilliam (1889) made another important observation on this subject He showed that, in animals, if the blood-supply to the heart be reduced by clamping one of the coronary arteries, the ventricles may fibrillate and, since ventricular fibrillation brings the circulation to a standstill, the animal dies Probably that is the reason why patients with angina pectoris are so liable to sudden death, their ventricles fibrillate on slight provocation, on account of the defective blood-supply

The enormous demand which the coronary circulation may be called upon to meet is shown by the observations of A V Hill (1927) on trained athletes He found that during severe physical exertion the output of the heart might be ten times what it is at rest, and he calculated that, to do this amount of work, the oxygen consumption of the heart-muscle alone must be as great as the total oxygen consumption of the whole body at rest It is not surprising, therefore, that any limitation of the coronary blood-flow will produce a corresponding limitation of the cardiac reserve

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CHAPTER VII

THE AETIOLOGY OF HEART DISEASE

THE facts and hypotheses upon which our present conception of heart disease rests are based on clinical and experimental observations dating from Harvey's discovery of the circulation of the blood These are the bricks of which the edifice of cardiology is built, but it is the manner in which the bricks are put together that gives a building its style and character to see its different features in their true perspective we must stand back and view it from afar Similarly, in medicine, it is only with the lapse of time that new facts and hypotheses assume their right proportions in the clinical picture The young clinician is apt to be carried away by enthusiasm for new 'discoveries' regarding which his more mature colleague prefers to keep an open mind

In the lifetime of the present generation the emphasis has twice shifted from one aspect of diagnosis to another, we can, so to speak, recognize three distinct schools of cardiac 'architecture' Up to the end of last century the architect was a morbid anatomist, whose aim it was to elicit, at the bedside, physical signs which would enable him to diagnose in his patients the structural lesions seen in the post-mortem room The most obvious of such signs were heart-murmurs, hence the importance attached to lesions of the heart valves

Just as Laennec's discovery of the stethoscope focused attention on auscultation, so the sphygmograph and the sphygmomanometer awakened interest in disorders of cardiac function, and clinicians began to pay more attention to symptoms and less to physical signs, the response of the heart to effort became the measure of its efficiency James Mackenzie

(see p 74) was the great exponent of this school His central theme was heart failure By carefully following up the after-histories of his cases Mackenzie convinced himself that murmurs were of little importance in prognosis, and that the expectation of life was worst, not in patients with loud murmurs, but in those who complained of shortness of breath or pain in the chest on exertion, those whose pulses were grossly irregular, and those who had oedema

Since the First World War our viewpoint has again shifted and, whereas for Mackenzie heart failure was the central feature, we now believe that the underlying cause of the heart failure is even more important, for each aetiological type of heart disease has its own natural history and, consequently, its own prognostic and therapeutic problems The significance of structural lesions and of disorders of function, on the other hand, is largely determined by the aetiological type of heart disease with which they happen to be associated

Take, for example, the relation of auricular fibrillation to congestive heart failure Mackenzie showed that fibrillation was often the cause, and not, as his predecessors believed, the result of heart failure It therefore took precedence in Mackenzie's view over all other features, save the heart failure itself, because it was regarded, and rightly so, by him as the precursor of that condition We now know that auricular fibrillation may be merely a transient disorder In patients with Graves disease, for example, when the thyrotoxicosis is controlled by subtotal thyroidectomy, normal rhythm may not only be restored, but permanently re estabished It is for that reason that the underlying cause of the disorder has come to be regarded as even more important than the dis order itself In Mackenzie's day the diagnosis would have been '*heart failure* resulting from auricular fibrillation' now it is '*thyrotoxic* auricular fibrillation with heart failure' To take another example, congestive heart failure, associated

with an attack of bronchitis, may disappear when the bronchitis subsides, whereas that which develops in the later stages of cardio-aortic syphilis is always progressive The important feature in these circumstances is the bronchitis or the syphilis, the heart failure is merely an episode in the course of the disease, in the one case transient, in the other terminal

Heart disease may be either congenital or acquired the latter is due to various causes which may be classified in three main groups—(1) inflammatory, (2) degenerative, (3) secondary to disease elsewhere Neoplasms of the heart are extremely rare

In an earlier chapter (p 4) I referred to the cardiac neuroses and to neuro-circulatory asthenia, with these we are not concerned here, since they are not due to disease of the heart, though they may be associated with it

CONGENITAL HEART DISEASE

The foetal circulation During intrauterine life oxygenated blood leaves the placenta through the umbilical vein, which opens into the left branch of the portal vein Most of this blood, however, short-circuits the liver by passing through the ductus venosus to the inferior vena cava Of the blood-stream entering the right atrium from this source, only about one-quarter goes to the right ventricle, the remaining three-quarters being diverted by the valve of the inferior vena cava through the foramen ovale to the left atrium (Fig 66), and thence to the left ventricle and ascending aorta, to supply chiefly the head, neck, and upper extremities The venous blood returning to the right atrium by the superior vena cava, on the other hand, follows the post-natal route and enters the right ventricle but instead of going to the lungs, which are still functionless, it is mostly diverted to the aorta through the ductus arteriosus to supply the trunk and lower

extremities, some returning to the placenta by the umbilical arteries

Oxygenated blood from the placenta mixes in the inferior vena cava with venous blood from the trunk and lower extremities, while blood in the aorta, distal to the ductus

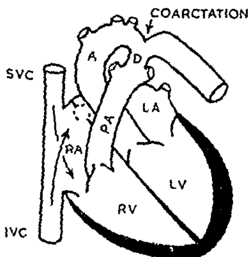


FIG 66 Diagram of foetal heart to illustrate course of circulation and common site of coarctation of aorta

SVC — superior vena cava *IVC* inferior vena cava *A* = aorta *D* ductus arteriosus *PA* = pulmonary artery *RA* = right atrium *LA* — left atrium *RV* right ventricle *LV* — left ventricle

arteriosus, is further deoxygenated owing to mixture with venous blood from the head and upper extremities Hence, in the foetus, the blood-supply to the trunk and lower limbs is less highly oxygenated than that to the head and upper limbs this accounts for the retarded development of the lower as compared with the upper parts of the body at birth When the umbilical cord is ligated, the lungs come into action and the blood from the right ventricle that previously entered the ductus arteriosus is aspirated to the lungs Finally, closure of the foramen ovale and of the ductus completes the transformation from foetal to post-natal conditions

Congenital abnormalities in the heart may be due to defective or arrested development in early foetal life, to persistence of foetal channels which normally cease to function after birth, or occasionally to intra-uterine infection, and recent work has shown that rubella, contracted by the mother in the early months of pregnancy, may give rise to congenital cardiac malformations in the foetus

When gross congenital anomalies are present, survival beyond infancy is rare There are, however, certain lesser defects, such as patency of the ductus arteriosus, patency of the atrial or ventricular septum, and coarctation of the aorta which, although they throw an additional burden on the heart, are compatible with the attainment of adult life, and may for many years produce little or no disability Mere curiosities, like congenital dextrocardia, do not affect the expectation of life

Not infrequently congenital defects are multiple, and this may make exact anatomical diagnosis extremely difficult

Cyanosis Some patients with congenital heart disease are cyanosed, others are not Cyanosis is an unfavourable sign Two factors may contribute to its production—mixing of the arterial and venous blood, and a deficient blood supply to the lungs This is well illustrated by 'Fallot's Tetralogy', the most common lesion in 'blue babies', in which pulmonary stenosis leads to hypertrophy of the right ventricle, and a right to left shunt is established through a defect in the ventricular septum, the aorta overriding the septum and communicating with both ventricles When there is a communication between the two sides of the heart, the blood normally flows from left to right, since the pressure in the systemic is higher than in the pulmonary circuit, and it is only when the pressure in the right side is higher than in the left that the lungs are short-circuited and the patient is cyanosed Estimation of the circulation time helps to confirm the presence of a right to left

shunt, for, when this exists, the arm-to-lung and arm-to-tongue times are approximately equal, while normally the latter is about double the former

The common types of shunt are a patent ductus arteriosus or a patency in either the atrial or the ventricular septum It is interesting to study the mechanical consequences of these three lesions In the case of the patent ductus, some of the blood from the aorta enters the pulmonary artery Hence the blood-flow through the lungs is increased and the left ventricle receives more blood than the right ventricle Conversely, in the case of an atrial septal defect, part of the blood which should have gone to the left ventricle is diverted to the right atrium and the right ventricle is overfilled at the expense of the left When the defect is in the ventricular septum, the right ventricle receives blood not only from the right atrium but also from the left ventricle, but, since the whole output of the right ventricle goes to the lungs and is returned to the left side of the heart, the left ventricle is overfilled to the same extent as the right Thus, if V be the normal output of the ventricle and S the blood-flow through the shunt, the effect of these three lesions may be expressed as follows

	Output		
	Right ventricle		*Left ventricle*
Patent ductus	$V-S$	$<$	$V+S$
Atrial septal defect	$V+S$	$>$	$V-S$
Ventricular septal defect	$V+S$	$=$	$V+S$

Catheterization of the heart has proved helpful in distinguishing between these three types of shunt, the catheter being introduced through a vein in the arm into the superior vena cava and pushed on, under fluoroscopic control, into the right atrium, the right ventricle, and finally the pulmonary artery This enables us to measure the oxygen saturation of the blood in these various situations If the oxygen saturation

[illegible] considerably higher than in the superior [illegible] is that oxygenated blood from [illegible] has been added to the venous blood in the [illegible] through a defect in the atrial septum A similar [illegible] between the blood in the right atrium and right [illegible] ventricular septal defect, while a higher [illegible] in the pulmonary artery than in the right ventricle [illegible]

[illegible] which have been made in recent years [illegible] in anaesthesia and in pre-operative and [illegible] have revolutionized the treatment [illegible] disease. Ligature of the patent ductus [illegible] on the left ventricle resulting from the [illegible] excision of the stenosed portion of the [illegible] the normal pressure relations [illegible] below the point of stenosis while, [illegible] anastomosis of the pulmonary [illegible] or with the aorta establishes [illegible] increases the circulation through [illegible] by raising the oxygen [illegible] As a life-saving measure th [illegible] are spectacular but suffi [illegible] to judge of the ultimate pro- [illegible] we are establishing communication [illegible] and the pulmonary circuits. This mu t [illegible] on the heart which may lead to heart [illegible] ligature of the patent ductus [illegible] As an alternative Brock and Campbell [illegible] on the stenosed orifice [illegible] heart lesions, in addition to [illegible] the ordinary routine clinical [illegible] examinations and from [illegible] additional evidence may be sought [illegible] (see p. 21). This permits visuali[illegible]

tion first of the right and then of the left side of the heart and recognition of abnormal communications between them

ACQUIRED HEART DISEASE

INFLAMMATION

Rheumatic, syphilitic, and streptococcal infections are the most important aetiological factors in this group, but pericarditis is sometimes tubercular, and acute lesions of the myocardium may result from diphtheria Other infections play only a minor part in the production of heart disease

Acute rheumatic carditis may involve the myocardium, the endocardium, and the pericardium, it often occurs in association with acute articular rheumatism or chorea but, in childhood, the heart rather than the joints may bear the brunt of the infection and, when articular and nervous symptoms are minimal, the condition is apt to escape recognition, fever, tachycardia, and anaemia being the only signs of infection

Valvular lesions are the most obvious manifestation in the chronic stage of rheumatic heart disease Where there is but slight damage these may give rise to no symptoms, mitral stenosis and aortic incompetence not infrequently being discovered in the course of a routine examination in patients unaware of any disability or of previous rheumatic infection Heart lesions indistinguishable from those due to rheumatic infection may follow scarlet fever

In *cardio-aortic syphilis* the site of election is the first part of the aorta Although often the resulting aortitis produces no disability and is only discovered at autopsy in a patient who has died from some other disease, the infection may spread towards the heart, producing incompetence of the aortic valve or obstruction of the mouths of the coronary arteries, a lesion to which I referred in an earlier chapter (p 103) as one of the causes of cardiac pain Aortic incompetence developing in a previously healthy middle-aged subject

is generally syphilitic A sequel of syphilitic infection now becoming less common, owing to more effective anti-syphilitic treatment, is aneurysm of the thoracic aorta

Clinically cardio-aortic syphilis remains latent for a long period and does not generally give rise to symptoms till ten or more years after the primary infection, by which time the damage is done hence treatment is essentially prophylactic (i e treatment of the primary infection)

The *streptococcus viridans*, a normal inhabitant of the mouth, is responsible for most cases of *subacute bacterial endocarditis* Prior to the introduction of penicillin, this was the most tragic of all forms of heart disease, for it often attacked young adults and there was no known therapeutic agent capable of arresting its progress To-day, provided diagnosis be not unduly delayed, the infection can be controlled in most cases, but unfortunately about a third of these patients die later from heart failure, which in some cases appears to be due to the mechanical effects of a progressive valvular stenosis A valve previously damaged by rheumatic infection, or a congenital malformation, is said to provide the most favourable nidus for the streptococcus viridans, but it is a curious fact that the underlying lesion is generally of a trivial nature I have never seen this disease arise in a patient with heart failure, and, in those with mitral lesions, a systolic is much commoner than a diastolic murmur This makes one wonder whether the valve really was damaged prior to its invasion by streptococcus viridans, for rheumatic infection generally produces stenosis

More acute types of bacterial endocarditis which run a very rapid course may be due to a haemolytic streptococcus, the gonococcus, the pneumococcus, or to other organisms Pneumococcal endocarditis is rarely diagnosed during life, as the cardiac manifestations are overshadowed by those of the associated pulmonary lesion

DEGENERATION

After the age of 40, heart disease is most often due to *coronary atherosclerosis* or to hypertension To the former I have already referred (p 102) in connexion with angina pectoris and coronary occlusion

High blood-pressure is a very common cause of ill-health in the second half of life Sometimes it is secondary to chronic Bright's disease or to other types of renal disease such as pyelo nephritis, but frequently it appears to arise spontaneously and is then called '*essential hypertension*' We still have much to learn about this condition, but the work of Goldblatt and his colleagues (1934) suggests that it may be due to renal ischaemia, which results in the secretion by the damaged kidney of a vaso constrictor substance 'renin' Further research on animals by Wilson and Byrom (1941) suggests that a vicious circle may be established, damage to one kidney causing hypertension which in turn, damages the vessels of the other kidney causing increased renal ischaemia, and so leading to a further rise in blood-pressure

Fortunately essential hypertension is a relatively benign condition, many patients remaining free from symptoms for years after hypertension is first discovered Some ultimately die from heart failure, others from cerebral haemorrhage '*Malignant hypertension*', in which the diastolic pressure may be very high, runs a much more rapid course, usually proving fatal within two years generally from renal failure It is probably merely a variant of the benign form, but tends to develop at a rather earlier age and manifests characteristic changes in the fundus occuli

'SECONDARY' HEART DISEASE

In view of the close partnership between heart and lungs it is not surprising that, in chronic pulmonary disorders such as emphysema, the strain on the right heart from increased

resistance in the pulmonary circuit should cause permanent damage, producing the so-called '*cor pulmonale*', and leading ultimately to heart failure

Thyrotoxicosis, especially in older subjects, is often associated with auricular fibrillation and heart failure, in fact in all cases of fibrillation which cannot be accounted for by rheumatic or degenerative heart disease, the possibility of thyrotoxicosis should be considered

Acute heart failure with oedema due to *vitamin B_1 deficiency* is occasionally seen in chronic alcoholics and is similar in aetiology to the heart failure of tropical beri-beri (Jones and Bramwell (1939))

Patients suffering from profound *anaemia* may complain of dyspnoea, oedema of the feet, and palpitation symptoms which, especially when associated with a systolic murmur and cardiac enlargement, are apt to lead to a mistaken diagnosis of heart disease Many years ago Balfour (1882) applied the term 'curable mitral insufficiency' to this condition

Whether the anginal pain of which patients with anaemia sometimes complain is entirely attributable to haemoglobin deficiency, or whether it occurs only when there is associated coronary disease, is still *sub judice*

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